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REPAIR OF POSTOPERATIVE MASTOID FISTULA

REPORT OF A METHOD

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A fistula in the wound from an operation on the mastoid is always a source of extreme annoyance to the patient and a great disappointment to the surgeon. Unsightly scars following certain types of operative procedures are unavoidable. Because the pathologic processes of the temporal bone are better understood than formerly, operative technic has been so improved that except in rare instances there is little need for the scar following a mastoid operation to be unsightly or for a fistula to be present. However, one should not hesitate to perform an adequate operation for fear of producing an unsightly scar. It is possible to perform an adequate operation and still not leave an unsightly scar.

In the early days of mastoidectomy postoperative fistulas developed because wounds purposely were packed in order to compel healing "from the bottom" to take place. Another reason often mentioned for this method of postoperative care was that it was undesirable for pockets containing septic foci to occur. It was customary to institute drainage instead of removing the diseased bone completely. At present, because of a general application of knowledge of the pathologic processes of the temporal bone, the surgeon is not content until satisfied that all the diseased bone has been removed. This makes it unnecessary to "pack the wound open" and permits closure of the edges of the wound. Most surgeons insist on approximation of the periosteum in addition to approximation of the skin and soft parts. With this method a satisfactory closure of the wound becomes a strong probability. The blood clot method of primary closure has been superseded by a modified blood clot method. General surgeons insist on careful avoidance of blood in wounds, because of the liability of infection ensuing in such a favorable medium.

At present, methods of closing mastoidectomy wounds so as to avoid the formation of fistulas are in general use. At least the upper three

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fourths of the wound is closed by sutures transfixing the periosteum, skin and soft parts, care being taken to approximate the edges without tension and to adjust the skin to direct apposition instead of permitting puckering. This closes the cavity over the tympanic antrum, the usual site of the formation of fistulas. The cavity of the mastoid is usually packed carefully with gauze, to control bleeding. The lower end of the gauze is extended through the lower part of the wound, so that it may be removed without disturbing the healed portion. Some surgeons prefer to use iodoform-impregnated gauze. This has a time-honored tradition in its favor, but too many patients are sensitive to iodoform to warrant its general use. Gauze impregnated with petrolatum has the advantage of not adhering to the underlying structures, and it controls the bleeding in a satisfactory manner. After the removal of the petrolatum gauze, drainage may be effected by means of a soft rubber tube. The sutures may be removed safely at any time after the fourth day. Attention to the aforementioned factors should prevent the occurrence of postoperative mastoid fistulas.

TYPES OF FISTULA

The types of fistula and the predisposing factors responsible for their formation have been described by Ashley.[1] Among the types encountered less frequently is a spontaneous fistula which occurs through erosion of the cortex and soft tissues overlying a diseased mastoid. Whether it is due to acute or to chronic infection of the mastoid cells, removal of the diseased tissue involved usually suffices in the treatment of a fistula of this type. The fistula following operation on the mastoid is the more common type and may vary from an opening 2 or 3 mm. in diameter in a shallow decompression to an opening 1 or 2 cm. in diameter at the apex of a large crater defect. Granulating tissue may or may not be present in fistulas of this type.

TREATMENT

Numerous procedures for closing persistent postauricular openings, whether following mastoidectomies or from other causes, have been described. The majority of the methods described in the literature are directed toward the restoration of a continuous postauricular cutaneous layer overlying the cavity that has resulted from radical mastoidectomy. A few pertain to the closure of postauricular fistulas following complete or simple mastoidectomy.

With few exceptions the literature on the subject of fistula behind the ear has appeared in the present century. As late as 1894 a textbook on diseases of the ear, by Dench,[2] made no reference to the treatment of

1. Ashley, R. E.: Postauricular fistula, Ann. Otol., Rhin. & Laryng. **46**:477-487 (June) 1937.

2. Dench, E. B.: Diseases of the Ear, New York, D. Appleton and Company, 1894.

postauricular fistula. In the simplest cases, those in which the opening is small, the plastic method, described by Hartmann,[3] of excision of the scar, freeing of the edges of the skin and suturing of the edges together over the opening is the one which would naturally appeal to every surgeon. For more complicated cases, more elaborate procedures are necessary. The Passow and Trautmann[4] method consists in circumscribing the opening about 4 mm. from the edge of the wound down to the bone posteriorly and down to the perichondrium anteriorly. This results in two crescentic flaps. These are inverted and the edges sutured. Finally, the skin over the denuded area is sutured. This method has proved popular for the closure of the small opening and is suitable for the fistula associated with the cavity that has resulted from mastoidectomy of the radical type.

In the von Mosetig-Moorhof[5] procedure a tongue-shaped flap of skin is loosened from the immediate neighboring parts and so cut that it is sufficient to cover the opening. An attachment of the U-shaped flap serves as an anchor for the rotated flap placed over the opening with its epithelial surface toward the cavity. Sutures are used to maintain placement. When a large flap is necessary a Thiersch graft is sometimes required to cover the area left exposed.

The methods of Passow and Trautmann and of Mosetig-Moorhof are given in detail, with illustrations, by Oppenheimer,[6] Kerrison,[7] Ballance,[8] Phillips[9] and Kopetzky.[10]

Alt[11] and Frey[12] advocated the use of paraffin for obliterating postauricular cavities and fistulas. This method is rarely if ever used today. Katz, Preysing and Blumenfeld[13] gave complete and well illustrated descriptions of the methods of Alt and Frey.

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3. Hartmann, A., cited by Ballance.[8]

4. Passow, K., and Trautmann, F., cited by Kopetzky.[10]

5. von Mosetig-Moorhof, A. R., cited by Kopetzky.[10]

6. Oppenheimer, S.: The Surgical Treatment of Chronic Suppuration of the Middle Ear and Mastoid, Philadelphia, P. Blakiston's Son & Co., 1906, pp. 386 and 389.

7. Kerrison, P. D.: Diseases of the Ear, ed. 4, Philadelphia, J. B. Lippincott Company, 1930, pp. 459-462.

8. Ballance, C. A.: Essays on the Surgery of the Temporal Bone, London, The Macmillan Company, 1919, vol. 1, pp. 228-253.

9. Phillips, W. C.: Diseases of the Ear, Nose and Throat: Medical and Surgical, ed. 7, Philadelphia, F. A. Davis Company, 1929, pp. 335-337.

10. Kopetzky, S. J.: Otologic Surgery, ed. 2, New York, Paul B. Hoeber, Inc., 1929, pp. 219-226.

11. Alt, F.: Ueber subcutane Paraffin-Injektionen, Monatschr. f. Ohrenh. **35:** 385-387 (Sept.) 1901.

12. Frey, cited by Katz, Preysing and Blumenfeld.[13]

13. Katz, L.; Preysing, H., and Blumenfeld, F.: Handbuch der speziellen Chirurgie des Ohres und der oberen Luftwege, Würzburg, Curt Kabitzsch, 1913, vol. 1, pt. 2, pp. 220-222; 1924, vol. 2.

Lermoyez and Mahn [14] employed a trapezium-shaped figure around the fistula, the skin being dissected well into the cavity in the shape of two wings. The two wings are turned inward so that they are brought into apposition with each other across the opening in the bone.

The introduction of the posterior relaxing incision resulted in many modified procedures for dealing with the fistulous area and for releasing the tension at the operative site by a suitable parallel posterior incision, with sufficient elevation of the flap. The procedures recommended by Kerrison, Goldstein,[15] Beck [16] and Kopetzky utilize the posterior incision to advantage. Some surgeons object to the large posterior scar that may result where a long incision is made.

Ballance used transplanted bone to fill the cavity frequently present. He first dissected the fistula around its outside margin, then sewed the edges of the fistula together, with the epithelial surface approximated. The bone graft, shaped to the cavity, was placed over this, and the edges of the skin were approximated over the bone. A secondary posterior incision, 2 or 3 cm. behind the original incision, served to relieve the tension.

Eagleton [17] used bone grafts and bone chips to obliterate the mastoidectomy cavity and advocated their use in selected cases.

In 1932 Straatsma and Peer [18] gave Eagleton credit for the proposal that fat grafts might be useful for filling postauricular cavities after complete mastoidectomy. They reported successful results in 15 cases. Fat fascia grafts are taken from the thigh or the abdomen. The skin is sutured over the grafts. The following year Bennett [19] reported the successful use of a fat graft.

During the last fifteen years many procedures utilizing flaps as fillers have been described. Kisch [20] obliterated the mastoidectomy cavity with a temporal muscle flap, left attached in the region of the zygoma. Almour [21] used a similar method but, in addition, rotated the muscle

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14. Lermoyez and Mahn, cited by Oppenheimer.[6]

15. Goldstein, M. A., cited by Ashley.[1]

16. Beck, J. C.: External Ear, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, pp. 417-418.

17. Eagleton, W. P.: The Reconstruction of the Mastoid Wound Cavity by the Use of Bone Grafts and Chips, Laryngoscope **29**:272-276 (May) 1919.

18. Straatsma, C. R., and Peer, L. A.: Repair of Postauricular Fistula by Means of a Free Fat Graft, Arch. Otolaryng. **15**:620-621 (April) 1932.

19. Bennett, A. B.: The Cure of Mastoid Fistula with Fat-Graft: Report of a Case, M. Ann. District of Columbia **2**:117-118 (May) 1933.

20. Kisch, H.: The Use of Temporal Muscle Grafts in Mastoid Operations, J. Laryng. & Otol. **43**:856-859 (Dec.) 1928.

21. Almour, R.: A Method for the Repair of Persistent Postauricular Openings, Laryngoscope **40**:799-801 (Nov.) 1930.

flap, so that the previous external surface faced the cavity and the periosteal surface faced externally. The method recently reported by O'Brien and Slaughter [22] also utilizes a temporal muscle flap in such a manner that the blood supply of the flap is not interfered with when the flap is in its new position in the postauricular defect.

Various types of so-called bridging operations have been described. In some of these the cavity is obliterated. In others it is not.

Lautenschläger's [23] method of secondary closure of retroauricular defects following radical mastoidectomy consists in uniting a short Körner flap to the freshened lower half of the posterior limb of the fistula and suturing the skin over it.

Watson [24] advocated the use of a crescent-shaped incision through the soft tissues down to the bone 3 or 4 cm. behind the fistula. The flap is then elevated from the incision to the fistula. The fistula is elevated behind and below and severed at its apex. A second flap is cut from the posterior meatal wall and sutured to the anterior margin of the fistula. The dissected fistula is then inverted, and the raw surfaces are approximated. Claus [25] and Claus and Blumenthal [26] used a periosteal bridge flap for covering the fistula after cleaning it. Popper [27] used a horseshoe-shaped periosteal flap cut from the tissue posterior to the original mastoidectomy incision. The flap is used to cover the bone and also fills the cavity. The method advocated by McNichols [28] to prevent the formation of a postauricular fistula following radical mastoidectomy has the advantage of permitting free movement of the skin over the elevated periosteal flap. According to the results obtained by Ashley, the operation devised by him is suitable for the closure of fistulas of all types. Two posterior incisions are used, a tongue-shaped muscular and periosteal flap being fashioned from the tissue between the first two incisions. This has the advantage that it does not involve an epithelial-covered surface in the flap used to fill the cavity.

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22. O'Brien, G. R., and Slaughter, W. B.: A Method for Reconstruction of Postauricular Defects, Arch. Otolaryng. **31**:65-73 (Jan.) 1940.

23. Lautenschläger, A.: Operations on the Ear; on the Nose and Its Appendages; Pharynx and Larynx; Trachea and Bronchi, in Kirschner, M.: Operative Surgery, Philadelphia, J. B. Lippincott Company, 1937, pp. 39-41.

24. Watson, D.: A New Operation for Closure of a Post-Operative Mastoid Fistula, Proc. Roy. Soc. Med. **23**:397-401 (Jan.) 1930.

25. Claus, G.: Brückenlappenplastik zur Behebung retroaurikulärer Defekte, Deutsche med. Wchnschr. **2**:1779-1780 (Oct. 16) 1931.

26. Claus and Blumenthal, cited by Engelmann, C.: Plastic Surgery of the Ear, in Kopetzky, S.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, p. 443.

27. Popper, O.: The Use of Periosteal Flap Grafts in Mastoid Operations, J. Laryng. & Otol. **47**:126-130 (Feb.) 1932.

28. McNichols, W. A.: Prevention of Postauricular Fistula by Periosteal and Subcutaneous Tissue Flaps, Ann. Otol., Rhin. & Laryng. **45**:475-480 (June) 1936.

It is surprising that more reports advocating the use of the pedicle flap did not appear in the literature prior to the report of Ersner and Myers.[29] They utilized a "racket-shaped" pedicle from the area just below the mastoidectomy wound and continuous with it. The pedicle is turned on its neck and placed in the cavity. The skin is sutured over it. A scalp flap method has been described by Straatsma.[30] An outlined scalp flap is measured to fit the cavity and taken so as to include the temporal artery at the base. The first step of the procedure consists in elevating the flap and covering the raw surface with a Thiersch graft. After two weeks the flap is turned down and fixed to the mastoid cavity. A heavy split graft taken from the thigh is used to cover the exposed area of the skull. Two weeks later the pedicle is severed and returned to its original place in the scalp and all the edges are approximated.

Other reports related to postauricular fistulas of cavities are those of Heine,[31] Campbell,[32] d'Onofrio,[33] Dawson,[34] Malan,[35] Tobeck,[36] Copps and McCormick,[37] and Wadsworth and Woodruff.[38] Worms [39] reported the successful closure of a postoperative mastoid fistula after the institution of antisyphilitic treatment.

A surgeon may be confronted with postoperative mastoid fistulas of various sizes and situations. The most troublesome from the standpoint of the patient are those situated in the upper portion of the mastoid and leading directly to the middle ear. In such a fistula the only closure

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29. Ersner, M. S., and Myers, D.: Variation of the Pedicle Flap for Epithelialization of the Radical Mastoidectomy Cavity, Arch. Otolaryng. **23**:469-474 (April) 1936.

30. Straatsma, C. R.: Repair of Postauricular Fistula Following Radical Mastoidectomy, Arch. Otolaryng. **19**:616-618 (May) 1934.

31. Heine, B., cited by Ashley.[1]

32. Campbell, A.: A Method of Applying Epithelial Grafts to the Mastoid Cavity, J. Laryng. & Otol. **38**:471-472, 1923.

33. d'Onofrio, F.: Le fistole della mastoide, Arch. ital. di otol. **41**:473-483 (Oct.) 1930.

34. Dawson, G. W.: Closure of Large Postauricular Fistulas and Depressions, Lancet **1**:1101 (May 11) 1935.

35. Malan, A.: Cicatrizzazione ritardata dopo interventi sulla mastoide, Valsalva **13**:67-69 (Feb.) 1937.

36. Tobeck, A.: Zur Wahl der Operationsmethode zum Verschluss von retroaurikulären Antrumfisteln, Hals-, Nasen- u. Ohrenarzt (pt. 1) **28**:227-231 (July) 1937.

37. Copps, L. A., and McCormick, G. L.: The Closure of Persistent Postoperative Mastoid Fistula with a Subcutaneous Pedunculated Flap, Arch. Otolaryng. **27**:472-473 (April) 1938.

38. Wadsworth, H. V., and Woodruff, G. H.: Retroauricular Fistula Following Mastoid Operations: Report of a Case Following Radical Mastoidectomy with Remarks on the Selection of Procedure, Illinois M. J. **76**:547-549 (Dec.) 1939.

39. Worms, G.: Fistule mastoïdienne postopératoire. Guérison par la traitement antisyphilitique, Ann. d. mal. de l'oreille, du larynx **47**:167 (Feb.) 1928.

possible by natural reparative processes is a thin layer of epithelial cells over the aditus. This thin scar breaks down easily and permits the secretion present in the ear to escape. This is especially true in cases of head cold. Forceful blowing of the nose may cause rupture of the thin scar over the aditus and escape of the mucopurulent discharge incident to the reinvolvement of the middle ear. To avoid the annoyance and inconvenience of such recurrent episodes the patient seeks relief.

The apparent explanation of the formation of postoperative mastoid fistulous tracts is that epithelial cells from the skin edges of the wound extend along the surface of the defect, which has been kept open by the prolonged use of packing. Under ordinary conditions of natural repair, the avoidance of prolonged packing of the mastoidectomy wound directed toward the tympanic antrum should prevent epithelized postoperative

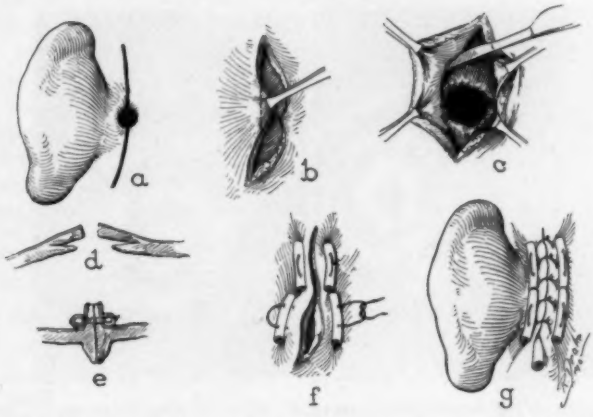

Fig. 1.—Method of repair of postoperative mastoid fistula. For further explanation, see text.

defects in a large measure. If the mastoidectomy has been thoroughly performed, there seems to be no need for packing of the wound in this manner. Mastoidectomy wounds do not heal from the bottom. Any surgeon who has been required to reopen the mastoid region in a subsequent infection unrelated to the original infection must have been struck with the fact that in the final healing a new tympanic antrum has formed. It is the opinion of one of us (H. I. L.) that this reformation results from perhaps two causes: (1) natural contraction of the granulation tissue and (2) the effect of air entering the middle ear and appendages through the eustachian tube. The repeated action of even the slight pressure resulting from the force of air may cause or increase the natural contraction associated with the healing process. If a defect in the tympanic membrane remains after the subsidence of the infection in the middle ear, the second factor described in the preceding

sentences will not operate. For the correction of such a postoperative defect one must employ a method of closure of the wound that does not permit the epithelial cells to extend along an open tract. Such a method requires the accurate approximation of freshly cut edges of the skin, so that healing by first intention will take place.

That repair of postoperative mastoid fistulas has presented a problem to numerous surgeons is evidenced by the considerable bibliography on the subject that has accumulated. Certain of the operative procedures described are formidable. The method we suggest seems simple by comparison. It has proved satisfactory in 12 consecutive cases and is presented for what it may be worth to other surgeons.

A Method of Repair.—It may not be possible adequately to repair a postoperative mastoid defect until bone has formed over any exposed

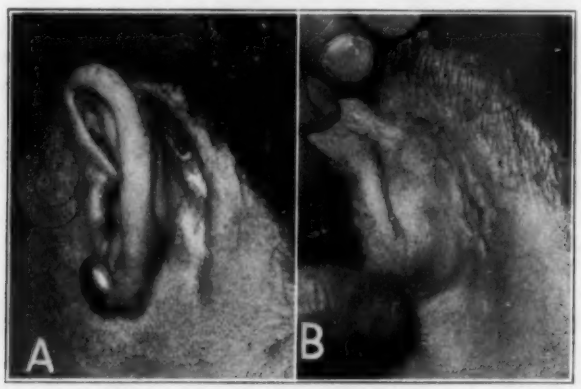

Fig. 2.—*A*, typical postoperative mastoid fistula of long standing. The deforming depression is evident. *B*, the repaired defect ten days after operation. At this stage the scar looks rough. The wound is closed tightly, and the depression has been overcome.

dura or sigmoid sinus. Only a single case has been encountered in which bone was not present. The time required for the bone to reform was approximately six months.

The steps in the operative procedure we suggest for closure of a postoperative mastoid defect can best be described by referring to figure 1.

The initial incision (*a*) is extended through the scar tissue above and below the fistula to points beyond which there is no depression. Then the lateral edges at the deepest point are incised down to the bone (*b*). The soft tissues are carefully separated from the bone in an outward direction, care being taken not to leave any epithelium beyond. If epithelium were to be left attached to bone, cholesteatoma might develop. It will be seen that there is an overabundance of soft tissue for closure but that the nature of the tissue is such that one could not expect healing to take place if the edges were approximated.

At this point the edges of the wound are undermined by an incision (*c*) into the soft parts deep enough and long enough so that a suitable edge for approximation is made available. The undermining incision is placed at ample distance from the edge to allow a free edge wide enough for approximation. It causes a V-shaped wound (*d*). There should be no tension or tendency to inversion. If any tendency to inversion is present, the incision is made longer or the underlying incision is carried deeper into the soft part until it is overcome. Trial approximation of the flaps is a good test for tension. In the trial approximation one notices that the lower lip of the V incision can be made to fill into the cavity of the V incision on the other edge of the wound (*e*). Thus, an overlapping subcutaneous closure is effected.

Accurate approximation is necessary to insure a good result. This may be accomplished by the type of tension or supporting suture illustrated (*f*). The particular advantage of this suture has proved to be that the edges of the wound are everted after they have been shaped by the cutting away of the excess of nonviable epithelial scar tissue. In addition, because the sutures (*g*) are not buried in swollen tissue, they are removed easily by cutting above the rubber tubing. The free edges of the wound are then closed with interrupted dermal sutures. A seton may be used at the lower end of the wound, to permit the escape of fluid from within. The dressing should be light, and it is not necessary to use pressure. The sutures are removed on the fourth day. There is need for surprisingly little postoperative attention. It has been our experience that the wounds were completely healed in ten days. Figure *2 A* shows a patient with a typical postoperative mastoid fistula. Figure *2 B* shows the same patient ten days after repair of the fistula.

SUMMARY AND CONCLUSIONS

A review of the literature pertaining to the surgical closure of postoperative mastoid fistulas is given. A method for repair is described by which an incision is used to undermine the edges of the wound, so that when these edges are approximated a wide surface for healing is made available. This method has been found successful in our hands in 12 consecutive cases in which there have been fistulas of various sizes. The use of an improved supporting suture, which has been found practical, is also described. The operation should not be undertaken until any defect in the bone overlying the dura or the sigmoid sinus has been filled in with newly formed bone. Care must be taken that epithelium is not permitted to remain in the depths of the wound.

FATE OF AUTOGENOUS SEPTAL CARTILAGE AFTER TRANSPLANTATION IN HUMAN TISSUES

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Otolaryngologists and plastic surgeons agree that septal cartilage is not as good a material for transplantation as rib cartilage. The general belief persists that septal cartilage is absorbed after transplantation and cannot be relied on as a material for filling saddle defects of the nose or to afford structural support for the nasal tip.

I have used autogenous septal cartilage grafts to correct small saddle depressions and to support the nasal tip in more than 500 cases and have failed to note gross absorption of the cartilage except when infection occurred after operation. Since my clinical experience differed from that of other plastic surgeons, I decided to check the accuracy of my observations by transplanting septal cartilage and removing it for microscopic examination at various intervals up to three years after transplantation.

CLINICAL IMPORTANCE OF SEPTAL CARTILAGE GRAFTS

Operations for displacement of the lower border of the septal cartilage utilize the cartilage as a free graft, partly or completely separated from the mucous membrane. The Metzenbaum operation,[1] Cohen's operation [2] and my operation [3] are examples of this type of procedure. If septal cartilage tends to degenerate after transplantation, a large percentage of failures should occur after these operations.

In submucous resection a surgeon frequently exposes the cartilage beneath the nasal bridge at a higher level than he desires for removal of the cartilage with the Ballanger swivel knife. In this manner a ridge of septal cartilage is exposed, free of its mucous membrane on each side. When the flaps are replaced, this ridge is essentially a free

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1. Metzenbaum, M.: Replacement of the Lower End of the Dislocated Septal Cartilage Versus Submucous Resection of the Dislocated End of the Septal Cartilage, Arch. Otolaryng. **9**:282 (Feb.) 1929.

2. Cohen, S.: Role of the Septum in Surgery of the Nasal Contour, Arch. Otolaryng. **30**:12 (July) 1939.

3. Peer, L. A.: An Operation to Repair Lateral Displacement of the Lower Border of the Septal Cartilage, Arch. Otolaryng. **25**:475 (April) 1937.

graft between the mucous membrane flaps. If such septal cartilage grafts failed to survive, there would be a large number of depressed noses.

Not infrequently in submucous operations one dislocates the beam of cartilage supporting the dorsum of the nose. The resourceful surgeon may select a segment of septal cartilage from his abundant supply and insert it beneath the skin overlying the depression, thus avoiding a postoperative deformity. Again septal cartilage is used as a grafting material.

The success of these operative procedures, as well as that of others not described, depends on the survival of septal cartilage transplanted between mucous membrane flaps or beneath the nasal skin. Therefore, a study of the fate of septal cartilage grafts after transplantation seems indicated.

EXPERIMENTAL WORK WITH LIVING CARTILAGE GRAFTS IN ANIMALS

The literature on experimental work with grafts of living cartilage in animals began with the contribution of Bert[4] in 1865 and has increased each year until the present. In this experimental work with animals three points of view as to the fate of adult cartilage transplants are expressed: 1. Cartilage tends to degenerate when transplanted.[5] 2. Cartilage tends to survive when transplanted.[4] 3. Cartilage survives when transplanted with its perichondrium but tends to degenerate and disappear when transplanted without its perichondrium.[6] I believe that some of these conflicting findings were due to the fact that different experimental animals were used by the investigators. In any event, one cannot accurately assume that human cartilage buried in human beings undergoes the same changes as animal cartilage buried in animals. The experimental work on animals, therefore, while interesting, does not decide the controversy as to the fate of cartilage buried in human beings.

PREVIOUS EXPERIMENTAL WORK WITH AUTOGENOUS COSTAL CARTILAGE GRAFTS IN HUMAN BEINGS

Koenig,[7] in 1896, was the first to use autogenous cartilage transplants in man, and since that time autogenous rib cartilage has been employed widely as a filling substance and as a structural support for soft tissues.

4. Bert. P.: Sur la greffe animal, Compt. rend. Acad. d. sc. **61**:587, 1865.

5. Ollier, L.: Traité expérimental et clinique de la régénération des os et de la production artificielle du tissu osseux, Paris, V. Masson & fils, 1867. Zahn, F. W.: Ueber das Schicksal der in den Organismus implantierten Gewebe, Virchows Arch. f. path. anat. **95**:369, 1884.

6. Fischer, E.: Ueber Transplantationen von organischem Material, Deutsche Ztschr. f. Chir. **17**:362, 1882.

7. Koenig, F.: Berl. klin. Wchnschr. **51**:1129, 1896.

There is, however, a great difference of opinion concerning the fate of autogenous costal cartilage following transplantation. In the literature one finds a variety of conflicting reports, all based on clinical observation of the grafted areas. Gillies [8] was the only investigator who actually removed a cartilage graft and examined it with the microscope. He stated that one specimen of autogenous rib cartilage examined microscopically eighteen months after transplantation showed no evidence of invasion or absorption and that the cartilage cells and the matrix appeared normal.

Desiring to obtain more definite information concerning the fate of grafts of human autogenous costal cartilage, I buried 9 segments of costal cartilage with perichondrium removed beneath the chest skin of 9 patients and 3 segments of costal cartilage without the perichondrium beneath the nasal skin of 3 patients.[9] The grafts were removed at intervals from six months to six years. Microscopic examination showed an absence of invasion or absorption of the grafts, and even in the six year section the cartilage cells and hyaline matrix appeared quite normal.

In this study 8 grafts were reported buried beneath the abdominal skin and 1 in the nose. Four have been removed and examined since that report was published. Two of these were buried beneath the nasal skin and removed twenty-eight months and five years, respectively, after transplantation. The other 2 were buried beneath the chest skin and removed nineteen months and four years, respectively, after transplantation. These 4 grafts appeared like normal living cartilage, with no evidence of invasion or absorption. From these findings one may conclude that autogenous costal cartilage grafts without perichondrium survive equally well whether transplanted beneath the nasal skin or beneath the chest skin.

Recently, I received autogenous costal cartilage grafts from Dr. Warren Davis and from Dr. Clair Straith. Dr. Davis' graft, with perichondrium on one side, had been inserted in the nose to support a deep saddle and was removed thirteen years after transplantation. The bulk of the cartilage showed no evidence of invasion or absorption, and the cells and matrix appeared normal. There was one rather small area at the edge of the graft, where fibrous tissue infiltrated the cartilage. This I interpreted as a fibrous replacement of a small portion of the cartilage injured at the time of transplantation. Dr. Straith's specimens represented two autogenous costal cartilage grafts with the perichondrium removed. One had been transplanted beneath the abdominal skin and removed five years and ten months after transplantation. The other

8. Gillies, H. O.: Plastic Surgery of the Face, London, Oxford University Press, 1920, pp. 13-14.

9. Peer, L. A.: The Fate of Living and Dead Cartilage Transplanted in Humans, Surg., Gynec. & Obst. **68**:603 (March) 1939.

had been transplanted beneath the nasal skin and removed five years after transplantation. There was no invasion or absorption of either graft, and the cartilage cells and hyaline matrix appeared normal.

On the basis of this experimental evidence, I believe that after transplantation in human beings autogenous rib cartilage without perichondrium survives as living cartilage and that it does not appreciably increase or decrease in size. Rib cartilage without perichondrium survives whether transplanted beneath the chest skin or beneath the nasal skin.

PREVIOUS EXPERIMENTAL WORK WITH AUTOGENOUS SEPTAL CARTILAGE GRAFTS IN HUMAN BEINGS

I am unable to find reference in the literature to any systematic or controlled experimental work with human autogenous septal cartilage. A large number of surgeons have used septal cartilage grafts clinically, and there is rather general agreement that septal cartilage is absorbed completely or reduced in size after transplantation.

With this opinion I disagree, on the basis of my own clinical experience with over 500 cases and the experimental evidence which follows.

EXPERIMENTAL PROCEDURE

All the work to be reported here was performed on human beings. The experimental subjects were patients who had extreme deflection of the septum associated with moderate saddle nose. After performing a submucous resection I inserted beneath the nasal skin sufficient septal cartilage to correct the saddle nose. The remaining cartilage was placed in a small pocket beneath the abdominal skin, to be used in case the cartilage inserted in the nose became infected. When the nasal operations were successful, I removed the septal cartilage from the abdominal pockets at intervals from two weeks to three years, for microscopic examination. Naturally, the perichondrium was removed from these grafts during the submucous operation.

The specimens were fixed in diluted solution of formaldehyde U. S. P., sectioned in the usual manner and stained with hematoxylin and eosin. Grossly, all of the specimens of transplanted cartilage appeared like normal septal cartilage and had about the same apparent size as before transplantation.

CONTROL SECTIONS

I examined stained sections of 15 segments of septal cartilage removed at submucous resection. These sections serve as controls in that they indicate the general condition of the cartilage grafts at the time of transplantation. From the examination of these control sections I made two important observations:

1. Eight of the 15 sections showed small indentations into the substance of the cartilage, due probably to penetration with the separator

during the submucous operation. Some of these nicks appeared deep, and a few sections showed V-shaped indentations, probably representing partial fractures of the septal cartilage.

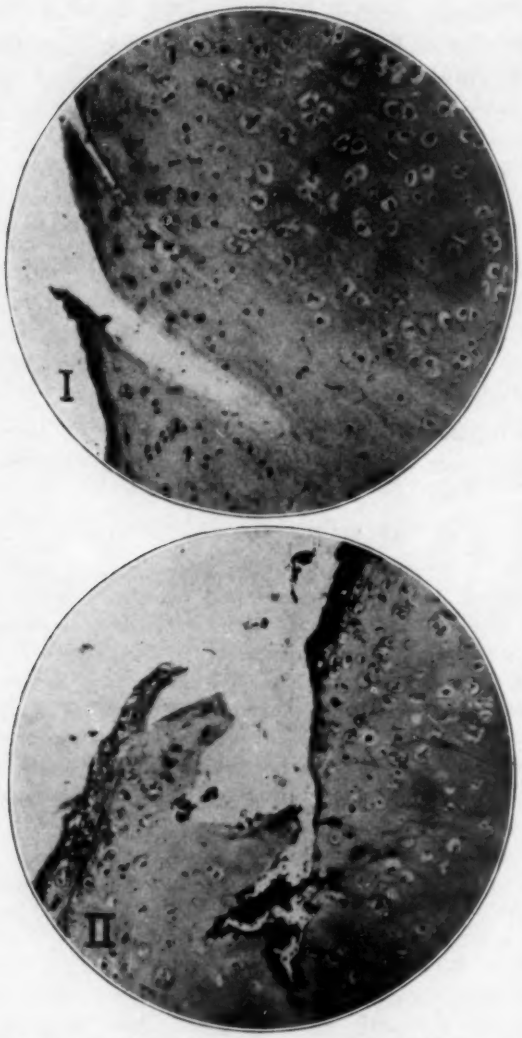

Fig. 1.—*I*, control section of septal cartilage removed in the usual manner at submucous resection. Note the two rather deep nicks in the cartilage, which were made by the separator during the operation. *II*, control section of septal cartilage removed at submucous resection. The magnification shows a deep indentation into the cartilage substance, with ragged edges. If this cartilage were used as a graft, host connective tissue would occupy the deep indentation and give the appearance of invasion.

2. In spite of attempted complete removal of all perichondrium, in 4 of the specimens small bits of this tissue were seen attached to the cartilage. Sometimes the strip of perichondrium was present in the form of a small flap attached to the cartilage at one end, with the other end free.

The presence of nicks or indentations in the control sections of cartilage is important to remember when one studies the buried cartilage grafts. Obviously, these nicks and indentations will be occupied by connective tissue after transplantation of the cartilage, and this gives the impression of invasion.

I studied the sections of buried septal cartilage with the control findings in mind, and I believe that the important facts to consider are the following ones:

If the penetration of some of the buried grafts by connective tissue represents invasion, it should be progressive, and in general older grafts should show a greater amount of invasion and a larger absorption of cartilage until finally the cartilage as a distinct tissue would disappear. If the grafts fail to survive transplantation, the cartilage cells also, which are the living elements of cartilage, should show progressive degenerative changes.

On the other hand, if the depressions in the cartilage represent small nicks or fractures made before transplantation, they should be present in some of the early grafts which have been buried only a short time as well as in those buried up to three years. If the grafts survive transplantation, the cartilage cells even in the oldest grafts should not show degenerative changes.

MICROSCOPIC EXAMINATION OF SEPTAL CARTILAGE GRAFTS

Eleven segments of septal cartilage obtained by submucous operations were transplanted beneath the abdominal skin of 8 adult patients. The grafts were removed for examination at intervals of two weeks, two months, three months, one year and fourteen days, twenty-seven months and three years.

The first 2 sections, buried two weeks, were buried in 2 different patients. A dense connective tissue capsule surrounds both grafts, in close contact with the cartilage except where separation occurred because of fixation. A moderate cellular reaction is present in the fat layer just outside the connective tissue capsule. This consists of a rather general infiltration of polymorphonuclear leukocytes and large and small mononuclear cells. There are also proliferation of fibroblasts and engorgement of blood vessels in the host tissue about the cartilage. A rather deep indentation is present in one graft and two shallow nicks in the other. These spaces are occupied completely by host connective tissue.

There is an absence of other cellular activity in the areas. [illegible]ita-tions correspond in depth and shape with some of those [illegible] the control sections of cartilage. The fact that a deep indentation [illegible]esent

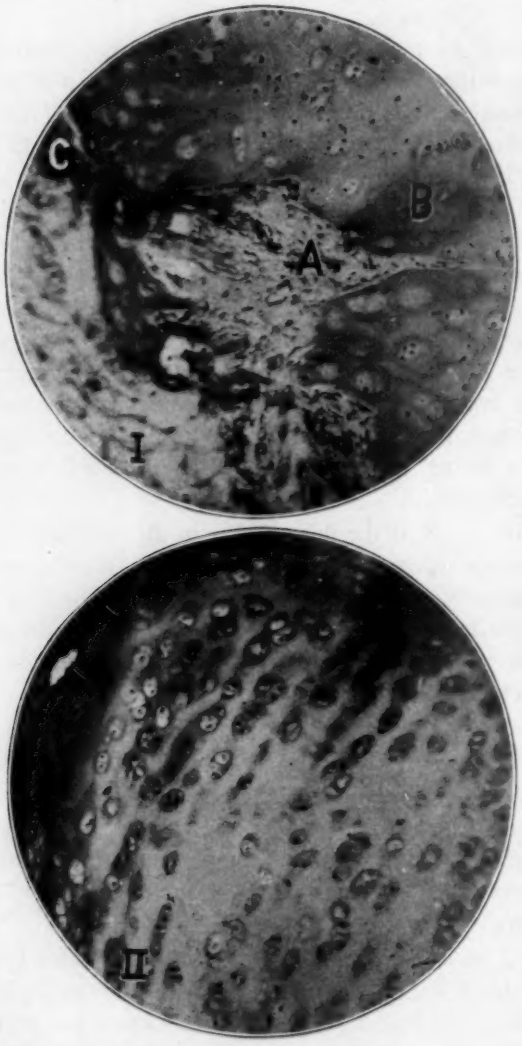

Fig. 2.—*I*, septal cartilage graft buried two weeks. A rather deep indentation, occupied by connective tissue, is present at *A*. There is an absence of cellular activity in the area, and the indentation resembles some of those found in the control sections. My belief is that this indentation was present when the cartilage was transplanted. The bulk of the cartilage graft is present, and the cells appear quite normal. *B* represents the cartilage; *C* represents the connective tissue capsule. *II*, septal cartilage graft buried three months. The cells and hyaline matrix appear normal, and there are no indentations in the graft.

in cartilage buried only two weeks leads one to believe that the indentation was present at the time of transplantation and does not represent invasion since the invasion of cartilage by host tissue is a slow process.

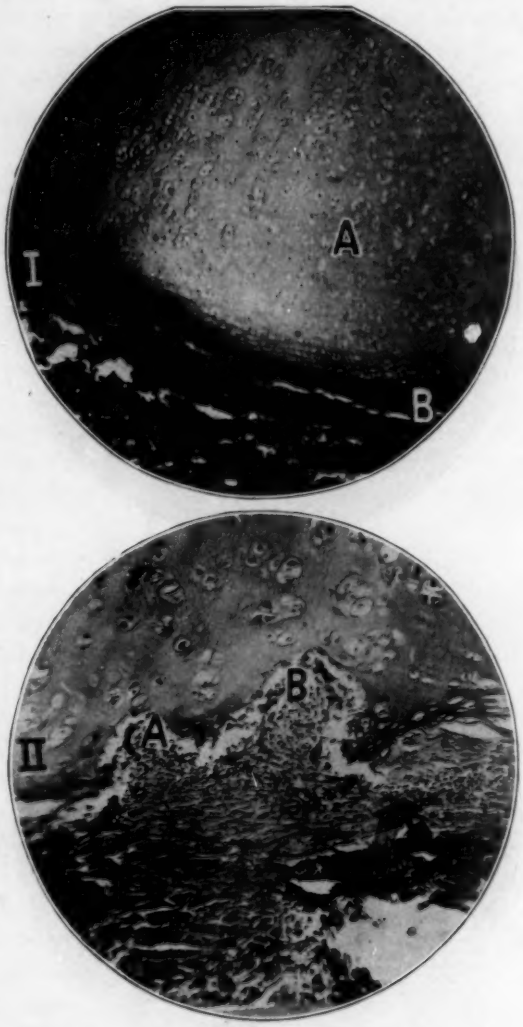

Fig. 3.—*I*, septal cartilage graft buried one year and fourteen days. *A* represents the cartilage and *B* the connective tissue capsule. There are no indentations in the graft, and the cartilage cells and matrix appear normal. *II*, septal cartilage graft buried twenty-seven months. Two shallow indentations (*A* and *B*) are occupied by connective tissue. The rest of the graft shows no indentations, and the cartilage cells and hyaline matrix stain normally. The shallow indentations correspond to those found in some of the control sections, and I believe that they were present in the cartilage at the time of transplantation.

(Invasion of dead preserved cartilage grafts did not occur until nine months after transplantation.[9]) On the whole, the cartilage cells appear quite normal, with dark staining nuclei and pale cytoplasm containing many fine filaments. The hyaline matrix also appears normal.

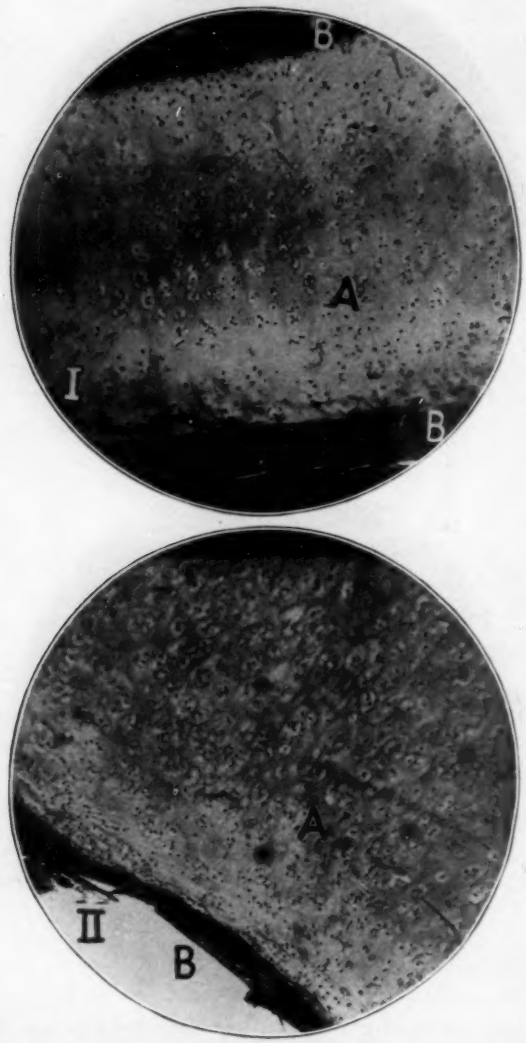

Fig. 4.—*I*, septal cartilage graft buried three years. *A* represents the cartilage and *B* the connective tissue capsule. Under high power magnification the cartilage cells appear normal and the hyaline matrix shows no degenerative changes. *II*, another septal cartilage graft buried three years. The cartilage is indicated by *A* and the connective tissue capsule by *B*. There are no indentations in the cartilage, and the cartilage cells and hyaline matrix appear normal.

In 1 specimen, buried two months, a connective tissue capsule surrounds the cartilage, and there is a general absence of reaction in the host connective tissue about the graft. No indentations are present in the cartilage, and the hyaline matrix and cells appear normal.

Two cartilage grafts buried three months in different patients show an absence of reaction in the surrounding connective tissue. A definite capsule surrounds each graft, and the cartilage cells and hyaline matrix appear normal. No indentations are present in the cartilage.

In the 2 cartilage grafts buried one year and fourteen days in the same patient, there is an absence of nicks. A connective tissue capsule envelops each graft, and the cartilage cells and hyaline matrix appear as normal living cartilage.

In the graft buried two years and three months, the bulk of the cartilage is intact and surrounded by a connective tissue capsule. There are two small indentations in the cartilage, which are occupied by host connective tissue, but there is no other cellular activity in this area and the edges of the indentations are smooth and do not show erosion. These indentations correspond in depth and shape to indentations found in some of the control sections. The cartilage cells stain exceptionally well, showing dark nuclei and clear cytoplasm. The hyaline matrix appears normal.

The oldest 3 grafts were buried in the same patient and removed three years after transplantation. Two of these are enveloped in the usual connective tissue capsules and show no indentations in the cartilage. The cartilage cells stain normally, and the hyaline matrix shows no degenerative changes. The third graft is also surrounded by a connective tissue capsule. There are, however, several indentations into the cartilage. These depressions are occupied by connective tissue and blood vessels without other cellular activity, and the sides of the indentations are smooth and do not show any signs of absorption. Two rather large blood vessels, surrounded by connective tissue, have grown into the graft. Dr. James Ewing interprets this as an effort on the part of the host tissue to retain the graft by supplying additional nourishment. He does not believe that it is part of an absorptive process. The hyaline matrix appears normal, and the cartilage cells show normal structure and staining reaction. In the host connective tissue just outside the cartilage are several small areas of new bone formation, but no calcification or other degenerative changes are seen within the cartilage graft.

All 3 of these grafts have about the same bulk as at the time of transplantation, and they appear in every way like normal living cartilage.

COMMENT

Eleven cartilage grafts which had been buried for various periods in human subjects show no larger number of indentations or deeper

Fig. 5.—*I*, a third septal cartilage graft buried three years. Two rather large blood vessels surrounded by connective tissue have grown into the graft. Dr. James Ewing interpreted this as indicating an effort on the part of the host tissue to retain the graft by supplying additional nourishment. He stated the belief that the penetration of blood vessels is not part of an absorptive process. Note the normal appearance of the cartilage about the blood vessels. The cartilage cells and hyaline matrix show no degenerative changes under high power magnification. *A* represents cartilage and *B* the connective tissue capsule. *II*, high power magnification, showing the normal appearance of cartilage cells and hyaline matrix in a septal cartilage graft buried three years.

indentations than were found in some control sections which were not transplanted. Indeed, the percentage of buried grafts showing indentations is less than that of the control sections. It seems reasonable to assume, therefore, that these indentations were present at the time of transplantation and do not represent areas of invasion.

This point of view is supported by the absence of cellular activity in the areas of indentation and by the smooth, noneroded appearance of the indentations. The fact that the deepest indentations were found in the 2 grafts buried two weeks rather than in the later grafts gives further support for this interpretation. Attention is also directed to the absence of degeneration in the hyaline matrix and to the normal appearance of the cartilage cells in all of the buried grafts.

All of these grafts possessed the same gross bulk when removed as when first transplanted, and if they had been used to fill a saddle of the nose or to support the nasal tip, they would have served as satisfactory material for this purpose.

Dr. James Ewing, consulting pathologist to the Memorial Hospital for the Treatment of Cancer and Allied Diseases, New York, examined sections of these grafts. He has permitted me to quote his opinion, as follows:

> The indentations in these cartilage grafts do not have the appearance of true invasion or absorption, and a comparison with the control sections indicates that the indentations were present in the cartilage at the time of transplantation. The general absence of degenerative changes in the hyaline matrix and the normal appearance of the cartilage cells demonstrate that these septal cartilage grafts have survived as living cartilage.

REASONS FOR CONDEMNATION OF SEPTAL CARTILAGE GRAFTS

I shall attempt to explain the probable reasons for the general condemnation of septal cartilage grafts. One explanation is the rather curious misconception regarding the structure of septal cartilage. I have found in the literature numerous statements by eminent otolaryngologists that septal cartilage is elastic cartilage or fibrocartilage and that for this reason it is absorbed shortly after transplantation. Septal cartilage is not elastic cartilage, and it is not fibrocartilage; *it is hyaline cartilage* and similar in structure to rib cartilage. If rib cartilage survives after transplantation, and I believe it does survive,[9] why should not septal cartilage also survive?

The nasal cavities are unsterile fields, and when septal cartilage is removed, it is contaminated by contact with the mucous membrane. For this reason septal cartilage grafts should always be inserted in healthy beds and firm pressure should be applied to prevent hematoma. If blood collects about the graft, separating it from direct contact with healthy tissue, infection of some degree is apt to follow. This infection will

cause rather rapid destruction of the graft and lead the surgeon to believe that the graft has been absorbed.

Another explanation is that the operator often selects too small a graft to fill a saddle defect in the nose. Nasal plastic procedures are usually done with the region under local anesthesia, and when the surgeon inserts a septal cartilage graft, he forgets that he has infiltrated both the tissue on which the graft rests and the overlying skin. Postoperative swelling conceals the result for some time, but when this subsides, imperfect correction is apparent. The surgeon then believes that the septal cartilage graft has been partly absorbed. If one makes a measurement of the depth of the saddle before injecting procaine hydro-

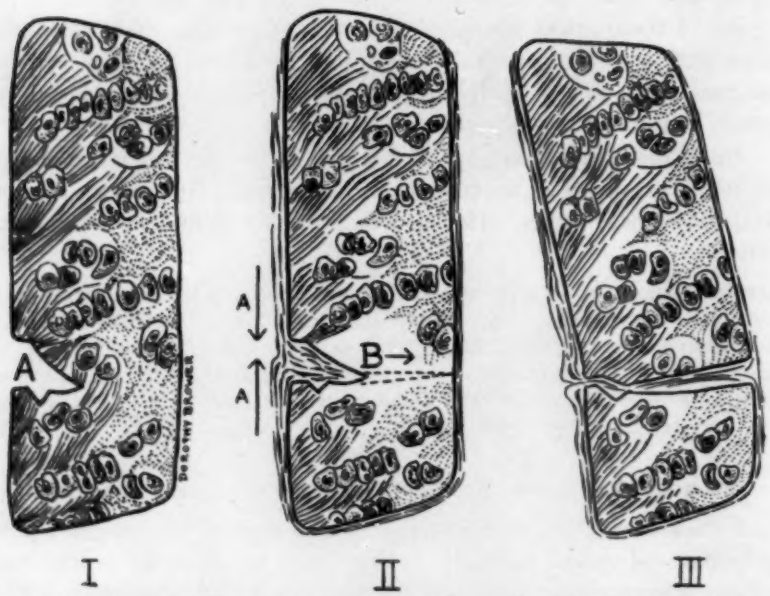

Fig. 6.—Diagrams illustrating how cartilage grafts with indentations may become distorted after transplantation. *I* represents a segment of septal cartilage removed at submucous resection. Note the deep indentation in the cartilage at *A*, made by the separator. *II* represents the same segment after transplantation. Connective tissue occupies the indentation and surrounds the cartilage graft. Contracture of the connective tissue capsule in the direction of the arrows, *A-A*, may split the cartilage as indicated at *B*. In *III*, connective tissue has grown completely through the split, or fracture, in the cartilage graft, and further contracture of the connective tissue capsule causes a mechanical distortion of the graft. No absorption of cartilage has taken place.

chloride and inserts a graft of adequate bulk, the result will be more satisfactory.

Correction of a deep saddle nose may require several thicknesses of septal cartilage sutured together. The operator should realize that the

segments of cartilage are not held in close apposition at all points by the suture and that after transplantation fibrous contracture will pull the segments together and reduce the bulk of the graft. In operations such as this, graft material should always be inserted in excess of the apparent requirement.

CLINICAL IMPORTANCE OF MICROSCOPIC FINDINGS

Microscopic examination of the septal cartilage grafts showed that indentations and cracks present in the cartilage before transplantation were occupied by connective tissue after transplantation. It would seem advisable, therefore, to select only smooth, even segments of cartilage for grafting purposes. The new bone formation in the connective tissue outside the three year graft demonstrates the bone-forming ability of connective tissue not related to periosteum. These small areas of bone represent only slight additions to the bulk of the graft and are not of clinical importance.

All of the septal cartilage grafts in this experiment had about the same bulk when removed as when first transplanted, and if they had been used to fill a saddle of the nose or to support the nasal tip, they would have served as satisfactory material for this purpose.

CONCLUSIONS

1. Septal cartilage is hyaline cartilage and similar in structure to rib cartilage.

2. Autogenous rib cartilage without perichondrium after transplantation in human beings, either beneath the nasal skin or beneath the chest skin, survives as living cartilage.

3. Autogenous septal cartilage without perichondrium after transplantation in human beings survives as living cartilage. It maintains the same gross bulk as when first transplanted and is not subject to absorption.

4. Only smooth segments of septal cartilage are suitable for grafting purposes. Nicks, indentations or fractures in the cartilage will be occupied by connective tissue after transplantation, and contracture of this connective tissue may distort or split the graft.

ELECTRICAL PHENOMENA OF THE COCHLEA IN MAN

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AND
T. J. CASE, M.D.
CHICAGO

In the last decade in otologic research a large literature has grown out of the study of cochlear potentials in experimental animals. Singularly few observations of the like electrical phenomenon in man have been reported. For several years we have been studying cochlear potentials in animals and in man in order to corroborate the observations of others and to throw more light on the relation of these potentials to hearing. It was also hoped that some clinical application could be found for an objective phenomenon associated with cochlear activity. However, for reasons described in this paper, human subjects suitable for such studies are difficult to obtain, and the work requires close cooperation between the clinical otologist and the neurophysiologist.

In 1935 Fromm, Nylen and Zotterman [1] obtained cochlear potentials in 2 subjects with perforated drum membranes. They used a five stage amplifying arrangement and a flexible electrode, placed against the promontory of the round window niche. The indifferent electrode was a metal bar held in the subject's mouth, the amplified potentials being led into a loud speaker. Frequencies from 200 to 5,000 cycles could be reproduced, but the intensities were too small to measure.

Between 1919 and 1938 Andreev, Arapova and Gersuni [2] reported that they were able to obtain cochlear potentials in man sufficiently large to record on the cathode ray oscillograph. In subjects with a large defect of the drum membrane, permitting the introduction of an electrode into the round window niche, sounds were introduced by means of a tube connected with a loud speaker. The equipment permitted the recording of disturbances above 1 microvolt. Twenty subjects were studied. When the ear was exposed to very loud sounds (near the threshold of pain), the magnitude of the potentials varied from 1 to 8

From the Division of Otolaryngology, the Division of Psychiatry and the Otho S. A. Sprague Memorial Institute, the University of Chicago.

1. Fromm, B.; Nylen, C. O., and Zotterman, Y.: Studies in the Mechanism of the Weber and Bray Effect, Acta oto-laryng. **22**:477-486, 1935.

2. Andreev, A. M.; Arapova, A. A., and Gersuni, A. V.: Bull. de biol. et de méd. expér. URSS **6**:495-496, 1938.

microvolts for frequencies from 500 to 2,500 cycles and from 5 to 26 microvolts for frequencies near 200 cycles. These investigators found that they could not obtain cochlear potentials when the loss of hearing exceeded 50 to 55 decibels or when there was much thickening of the mucous membrane of the middle ear. The largest cochlear potentials were obtained when the electrode was in the round window niche, and a shift of even a few millimeters in the electrode resulted in a marked reduction in the potentials.

In several subjects a correlation was made between the intensity of the sound stimulus and the magnitude of the potential. These studies indicated that the magnitudes of the electrical potential reach their peak before the limit of intensity of the sound stimulus and subjective loudness has been reached. The character of the electrical potentials obtained in man generally resembled those obtained in animals except for a striking difference in magnitude. In contrast to the small potentials, 1 to 25 microvolts, obtained in man, with the same experimental technic potential magnitudes approaching 1,000 microvolts were obtained in animals.

Our equipment for studying cochlear potentials consisted of an amplifier and a loud speaker or head phones. Sound was delivered to the ear by means of a garden hose leading from a loud speaker driven by an audio-oscillator or by means of the spoken voice and by whistling. When any electric sound producer was used, proper precautions for shielding were taken, to avoid microphonic and electrical artefacts. When the potentials were large enough in comparison with the noise level of the amplifier, they were visualized with a cathode ray oscillograph and photographed by means of a mirror oscillograph. Determinations of intensity were made by comparison with a signal passed through a calibrated attenuator from a controlled audio-oscillator source. A large number of negative results in human subjects in contrast to consistently positive responses in animals (rabbits) first led us to investigate the potentials in monkeys (Macacus rhesus). This was accomplished by using a firm insulated copper wire for contact with the cochlea and a metal clip on the lobe of the ear as the ground electrode. The stiff copper wire could be handled as a probe and passed under direct vision along the external auditory canal and through the drum membrane, the bared end of the wire being brought to rest against the promontory in the region of the round window niche. Good cochlear potentials were obtained regularly with the monkeys under anesthesia induced with pentobarbital sodium or vinethene. Furthermore, the cochlear potentials were not diminished in unanesthetized monkeys (fig. 1). A low conversational voice at the monkey's ear could be understood easily at the loud speaker.

For the human subjects, the active electrode consisted of a hair-thin enamel-insulated copper wire about 15 cm. in length, the insulation being removed for a centimeter from both ends. The bared wire of one end was wound around a cotton pellet of pinhead size. This cotton was dipped in Ringer's solution, carried into the ear with the attached wire by means of a fine ear forceps and placed where desired under direct vision. No harmful effect was observed from this procedure. The other end of the wire was connected by a metal clip with a shielded cable leading to the input stage of the amplifier. The indifferent electrode was a silver disk fastened to the skin over the mastoid with electrode paste. Usually, the subject was placed on a cot in an electrically shielded cubicle.

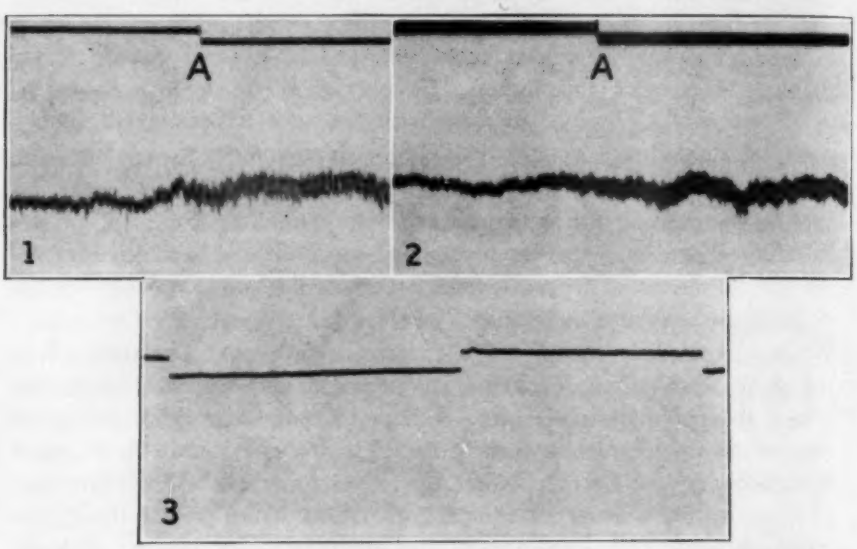

Fig. 1.—Records of cochlear potentials in a monkey for a tone of 300 cycles of moderate intensity (probably below the threshold of the middle ear muscles). *1* was made with the monkey under vinethene anesthesia and *2* without anesthesia. To the left of *A* the tube conducting the sound is away from the ear, at *A* it is rapidly brought near the ear, and to the right of *A* it remains near the ear. *3*, calibration: displacement for a 30 microvolt signal.

The loud speaker or head phones driven by the amplified cochlear potentials were placed in an adjoining room, where responses to the spoken voice and other stimuli were listened for. A group of subjects with normal hearing, when tested with the electrode against the tympanic membrane, gave negative results. The large number of negative results obtained with the electrode in the round window niche was found to be due principally to defective hearing in the ears examined. Most subjects with a large defect in the drum membrane have associated lesions of the

conduction mechanism producing a 40 or 50 decibel drop in the threshold curve. The subjects giving positive results were all found to have good function in the tested ear (less than a 20 decibel drop in their threshold curves). Obviously only a few subjects were to be found with good hearing in the presence of a large permanent defect in the drum membrane. In addition to this group, there were several subjects with good function from whom positive results were obtained who had a markedly thinned retracted drum membrane adherent to the promontory so as to skeletonize the torus of the round window niche. In those, the electrode was placed in the region of the round window niche, against the adherent drum membrane. It was found that in order to get consistent responses the area had to be cleaned well of any film of cerumen, since such a film appeared to insulate the cochlear potentials from the cotton wick electrode. The best potentials were obtained when the electrode was in the round window niche, and a shift of the electrode of even a few millimeters from this point resulted in a marked reduction or in disappearance of the cochlear response. One subject with excellent auditory function and a dry anterior-superior perforation was an exception to the rule. In testing this subject the electrode was wedged between the perforated drum and the cochlear capsule in the region of the apex of the cochlea. Consistently good cochlear potentials were obtained on three different occasions. A loose contact and drying of the cotton were other factors causing negative results.

REPORT OF EXPERIMENTS

CASE 1.—W. O., a boy aged 12, had a marked retraction of the right tympanic membrane, which was adherent to the promontory, skeletonizing the torus of the round window niche. His hearing was excellent, the audiogram showing only a 10 to 20 decibel drop for all frequencies tested. With the electrode against the drum in the region of the round window niche, responses to the spoken voice and to pure frequencies were clearly heard on several different occasions. The response was loudest for 1000 cycles, but the background noise of the amplifier prevented visualization on the screen of the cathode ray oscillograph even when a 1000 cycle band pass filter was used. The impression obtained by listening to the loud speaker was that the maximum output (maximum loudness) occurred before the maximum intensity of the stimulating sound was reached.

CASE 2.—R. O., a boy aged 14, had bilateral chronic quiescent otitis media. A defect in the right drum membrane exposed the round window niche. Hearing was depressed least for 2048 cycles (15 decibels), and constant responses were obtained for frequencies around this level. Slight displacement of the electrode from the round window niche abolished the response.

CASE 3.—L. W., a man aged 31, had a moderately large defect of the drum membrane, which exposed the round window area, but excellent function was retained. The mucous membrane of the middle ear was practically normal in color and thickness. Clear cochlear responses to the spoken voice and pure frequencies were obtained with the electrode in the round window niche. While these responses could be heard clearly in the loud speaker through the noise of the amplifier, they could

not be visualized with the cathode ray oscillograph. A quantitative measure of the cochlear response was made by matching the loudness of the response to 800 cycles at the maximum output of the oscillator with the loudness of a known controlled input signal of 800 cycles. This measurement indicated an intensity to the cochlear responses of about 3 microvolts. At another examination, responses to a whistle of about 500 cycles could be not only clearly heard but seen on the screen of a cathode ray oscillograph. For a loud whistle the displacement of about 5 mm. on the screen represented about a 12 microvolt input of cochlear potentials.

CASE 4.—J. L., a youth aged 18, had a marked thinning of the left drum membrane, which was adherent to the promontory so as to skeletonize the torus of the round window niche. Auditory function was very good, with only a 10 decibel drop in the threshold curve. On several occasions cochlear potentials were obtained after a thin film of cerumen had been removed from the surface of the drum membrane by irrigation. Moving the electrode away from the drum membrane abolished the response.

CASE 5.—In Miss G., aged 35, the left drum was perforated to expose the round window niche. After an acute infection had subsided, the mucous membrane over

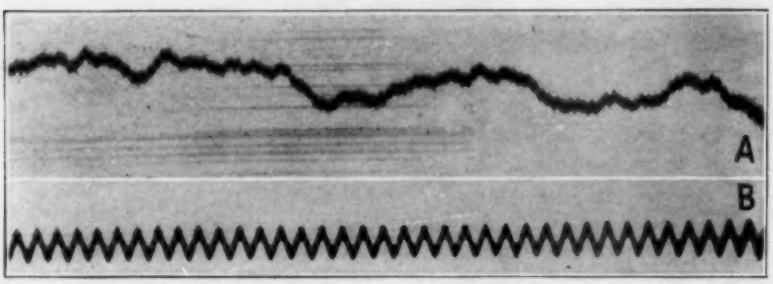

Fig. 2.—Record of cochlear potentials in the human subject for a tone of 2,700 cycles. *A* represents the physiologic tract. On the slow undulations of the base line is seen the fine regularly recurring spikes of 2,700 cycle frequency. This is recognizable by measuring against the 1,000 cycle timing wave at *B*.

the promontory was thin and function was good. On three different occasions cochlear potentials were obtained. The loudest response obtained was for 2700 cycles, at which frequency the resonance of the tubing amplified the sound output. The cochlear response was recorded along with a 1000 cycle timing wave (fig. 2). On one occasion, when ear phones were used instead of a loud speaker, the signal for a 1000 cycle tone was very clear. Nevertheless, this could not be identified on the screen of the cathode ray tube or in the record made with the mirror oscillograph. These observations indicate the superior ability of the observer's ear to discriminate between the background noise of the amplifier and the physiologic response. The nonlinear characteristics of the head phone (peaking in the neighborhood of 1000 cycles) was a factor in obtaining a loud cochlear response for a sound of this frequency.

CASE 6.—Miss F. G., a girl aged 13, had a dry anterior-superior perforation of the right drum membrane, exposing the apical area of the cochlear capsule near the tensor tympani muscle. The function was practically normal. On three occasions very good responses were heard with the electrode placed against the capsule

through the perforation. The spoken voice and the whistle, particularly in the lower frequencies, were heard but could not be recorded.

CASE 7.—S. B., a man aged 32, had a posterior-inferior perforation of the drum membrane, exposing the round window area. The function was good. There was little swelling of the mucous membranes of the middle ear. Good responses to whistling were heard with head phones and loud speaker, but they could not be recorded.

CASE 8.—M. C., an unmarried woman aged 30, had an old dry perforation of the drum, about 2 mm. in diameter, which exposed the promontory near the round window niche. Jacobson's nerve and associated blood vessels could be seen through the normal pale thin mucous membrane of the promontory. Function was excellent. An electrode was introduced through the perforation to rest against the promontory posterior to Jacobson's nerve, near the torus of the round window niche. Clear and loud responses for whistling through an octave of frequencies from about 500 to 1000 cycles were heard in the telephone receiver. The spoken voice, however, could not be heard. Again the background noise was too great to permit visualization with the cathode ray oscillograph. Removing the electrode from the middle ear and allowing it to rest against the epithelium of the external auditory canal abolished the response.

NOTE.—To eliminate a large part of the background noise due to electrical disturbances between the two electrodes, it may be possible to devise a concentric electrode that can be used in the human ear.

CASE 9.—R. B., a youth aged 16, claimed that he had been totally deaf for the past seven months. He stated that hearing impairment had begun six years before, after an attack of scarlet fever and mastoiditis on the right side. Transient attacks of blindness and loss of the senses of taste and smell were also noted by the patient. His ear drums were normal, and careful tests, together with observation in the hospital, suggested the diagnosis of hysteria. Nevertheless, his reactions had led his family and even several otologists to believe that he was totally and permanently deaf. It was decided that the recovery of cochlear potentials would add objective evidence to the clinical impression that the illness was functional. Furthermore, the subject was given the impression that the procedure would help to restore his hearing. With the patient under anesthesia induced with avertin with amylene hydrate supplemented by vinethene, the right drum membrane was incised over the posterior inferior quadrant, and a cotton ball electrode was inserted into the round window niche through the opening in the drum membrane. Clear responses to a low and to a moderate spoken voice at the ear and also to whistling were obtained, as well as responses to pure frequencies, such as 500 and 1000 cycles. However, even with the use of a band pass filter it was impossible to visualize on the cathode ray screen the response for a tone of 1000 or 500 cycles. The responses appeared clearer and louder to the observer's ear than any previously obtained. Before the examination was concluded, the electrode was pulled out of the middle ear and allowed to rest against the drum. No cochlear response could then be obtained.

NOTE.—While an anesthetic is needed, the actual contact with the cochlea may be made by a firm wire electrode piercing the drum, as in the experiments on monkeys, and need not require myringotomy.

Psychiatric guidance of this patient during the postoperative period was associated with an immediate return of his sense of taste and of smell and a return of hearing within twenty-four hours. It was then possible to make an audiogram, and

it was found that he had a normal threshold curve. The ear which was operated on regained its normal appearance in three days. The patient left the hospital freed of his hysterical symptoms.

ELECTRICAL STIMULATION OF THE COCHLEA

Another series of observations included in this report pertains to the electrical stimulation of the cochlea. It has been known for many years that a subject perceives sound when an oscillating current of audio-frequency is passed through the ear. This phenomenon was studied in detail by Arapova and Gersuni,[3] Stevens [4] and Clark Jones, Stevens and Lurie.[5] We corroborated the findings of these observers that the threshold of pain was reached before the auditory sensation became loud, that the sound was thin and metallic (contained harmonics) in spite of the purity of the wave form introduced and that the pitch did not always correspond to the frequencies of the oscillating current, often being an octave above and described by 1 patient as an octave below. The ear was stimulated by filling the external auditory canal with a saline solution, in which the bared end of a copper wire electrode was immersed. An electrode with a cotton pledget soaked in saline solution placed against the drum or promontory was equally effective. The other electrode was a silver disk held by electrode paste to the skin over the mastoid or tragus. These electrodes were connected to the output of an audio-oscillator. The limited range between the threshold of hearing and the threshold of pain was true for subjects with normal ears as well as for those with a destroyed drum membrane, in which the saline solution came directly in contact with the promontory and windows of the labyrinth. In the subjects with a destroyed drum membrane, contraction of the facial muscles from electrical stimulation of the facial nerve also limited the intensity of the stimulus that could be tolerated. More current was needed for a theshold response in a normal ear than in an ear in which the middle ear mechanism had been destroyed. On the other hand, in subjects with only a moderate degree of nerve deafness the threshold of pain was often reached before an auditory sensation was elicited. Arapova and Gersuni [3] reported that they could increase the loudness and clearness of the auditory sensation by passing a direct current through the ear along with the oscillating current. In an attempt to get a larger electrical stimulus to the cochlea than could

3. Arapova, A., and Gersuni, G.: On the Frequency of Alternating Current and the Pitch of the Tone During Electrical Stimulation in the Auditory Apparatus, Tech. Phys. URSS **5**:447-462, 1938.

4. Stevens, S. S.: On Hearing by Electrical Stimulation, J. Acoust. Soc. America **8**:191-195, 1937.

5. Clark Jones, R.; Stevens, S. S., and Lurie, M. H.: Three Mechanisms of Hearing by Electrical Stimulation, J. Acoust. Soc. America **12**:281-290, 1940.

be tolerated by a human subject, further observations were made on a rabbit under anesthesia induced with ethyl carbamate. Both bullae were opened, and one electrode was introduced into one round window niche, while the other electrode was clipped to the auricle. The cochlea was stimulated with alternating currents at audiofrequencies from an audio-oscillator, and the opposite ear was observed for reflex contractions of the middle ear muscles. While the muscles were seen to contract briskly to normal sound stimuli of only moderate intensity applied to the opposite ear, no contractions to electrical stimulation of the cochlea were observed even when the strength of the current passing through the cochlea caused violent contractions of the face and neck muscles on the side stimulated. This indicated the weakness of the cochlear stimulus electrically induced as contrasted to the normally induced (acoustic) cochlear action. On the other hand, the cochlear response from the ear for air-conducted sounds was heard clearly in the loud speaker.

The limited range in the loudness of the auditory sensations obtainable by passing oscillating currents through the ear as contrasted with the ability of the ear to respond to normal sound stimuli of enormous range may be related to the small and limited range of the cochlear potentials in man (even to large sound stimuli approaching the threshold of pain). As Davis [6] suggested, possibly the structures in the cochlea driven by an oscillating current and leading to auditory sensations, particularly in the absence of a conducting mechanism, are the same ones that respond to sound stimuli by setting up electrical disturbances that we pick up as cochlear potentials. Our observations corroborate those of Andreev, Arapova and Gersuni [2] in human beings and of Stevens and Davis [7] in animals that the magnitude of the cochlear potentials reaches a maximum, above which it cannot be carried by an increase in the intensity of the auditory stimulus.

In a number of investigations of the origin of cochlear potentials in experimental animals and their relation to normal cochlear function, the findings have favored the impression that structures (Reisner's membrane) other than those related to the normally activated neural elements (hair cells) may be responsible for cochlear potentials.[8] Although experiments can be devised to show this probable dissociation between cochlear potentials and normal cochlear function, it is likely that these situations rarely prevail as clinical and pathologic entities

6. Davis, H.: The Clarification of Certain Phases of the Physiology of Hearing, Laryngoscope **50**:747-755 (Aug.) 1940.

7. Stevens, S. S., and Davis, H.: Psychophysiological Acoustics: Pitch and Loudness, J. Acoust. Soc. America **8**:1-13 (July) 1936.

8. Hallpike, C. S., and Rawdon-Smith, A. F.: The Wever and Bray Phenomenon—A Summary of the Data Concerning the Origin of the Cochlear Effect, Ann. Otol., Rhin. & Laryng. **46**:976-990 (Dec.) 1937.

(i. e., degeneration of the spiral ganglion with normal blood supply). Should cochlear potentials be obtained in man when no function can be detected clinically, some such neural degeneration would be suggested. Our experiences, particularly in case 9, together with those of Andreev, Arapova and Gersuni, suggest that when cochlear potentials can be detected in man good cochlear function probably exists. A possible clinical use for such an objective cochlear phenomenon in cases of hysterical or functional deafness suggests itself. The phenomenon can be employed as an objective test of cochlear activity that may help to confirm the diagnosis and promote by suggestion the disappearance of symptoms. Further observations on these electrical phenomena in man seem indicated, both to increase fundamental knowledge of the auditory mechanism and in the hope that a greater clinical application may be found for the phenomena.

CONCLUSIONS

1. Cochlear potentials can be obtained regularly in man when there is a normal or nearly normal auditory threshold curve.

2. The potentials are only a few microvolts in intensity but can be clearly detected in a loud speaker or head phones.

3. Cochlear potentials reach a maximum, above which they cannot be carried by an increase in the intensity (loudness) of the auditory stimulus.

4. The limited range and intensity of cochlear potentials may be related to the limited range and intensity of the auditory sensation arising from electrical stimulation of the cochlea and suggest a common locus of stimulation.

5. One should think of the cochlear potential not as synonymous with the physiologic reaction of the organ of Corti or with auditory sensation but rather as parallel to them.

6. The determination of cochlear potentials in man may be useful in the diagnosis of functional or hysterical deafness.

MODERN TREATMENT OF ACUTE TONSILLITIS BY INJECTION OF BISMUTH COMPOUNDS

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Seven years ago I published the first acticle [1] on the brilliant results obtained in the treatment of acute lacunar tonsillitis by injection of bismuth compounds. Since then, in a series of papers published in Brazil and abroad I have pointed out the advantages of this new treatment over the other forms of therapy.

In an analysis of forty-six papers I have found that my original work has been corroborated and the facts verified. I shall here review some of the recent works of various observers in this field.

Pain is the most evident symptom of acute tonsillitis and the one causing the patient most distress. Bismuth has a rapid effect on this symptom, and it has been observed that in eight to ten hours after injection of a bismuth compound swallowing has become painless and the patient feels better. Both Errecart,[2] in Buenos Aires, Argentina, and Nuernbergk,[3] in Breslau, Germany, stated that in some patients they have observed relief from pain in four hours. Rojas and Nijensohn [4] reported prompt relief of pain following injections of bismuth compounds in more than 100 cases.

Most patients with acute tonsillitis have general malaise and a rise in temperature. Bismuth has been found to reduce the temperature to the normal within twenty-four hours. Stiehr [5] reported that of 152 patients 38 per cent experienced a rise in temperature to between 39 and 40 C. (102.2 and 104 F.) and that under bismuth therapy the

From the Department of Otology, Rhinology and Laryngology of the University of Brazil.

1. Monteiro, A.: Une nouvelle thérapeutique. Le bismuth dans le traitement des angines aiguës non spécifiques, Ann. d'oto-laryng., 1934, p. 557.

2. Errecart, P. L.: La bismutoterapía en las anginas agudas no especificas, Rev. argent. de oto-rino-laring. **3**:310, 1934.

3. Nuernbergk, W.: Ueber die Wismuttherapie der unspezifischen Angine, Hals-, Nasen- u. Ohrenarzt (pt. 1) **27**:384, 1936.

4. Rojas, F. A., and Nijensohn, M.: El bismuto en el tratamiento de las anginas agudas no especificas en los niños, Arch. Assoc. méd. d. Hosp. Salabery **1**:3, 1935.

5. Stiehr: Erfahrungen bei der Behandlung eitrige Anginen mit einem Wismutpräparat (Etosio), München. med. Wchnschr. **82**:1761, 1935.

temperature fell to normal within twelve to twenty-four hours. Köhler [6] stated that the earlier in the course of the disease the bismuth is given the sharper is the drop in temperature. Chanine [7] also emphasized the importance of administering the drug early in cases of acute tonsillitis.

The action of bismuth can be seen rather promptly in its effect on the local tissue and the cervical lymph nodes. In the majority of cases the follicular exudate disappears within the first twenty-four hours. At this stage the tonsils appear red, as in any acute infection. It has been inferred that the etiologic agent in these cases is Streptococcus and that this organism is the only one which responds to bismuth therapy. Exceptions to this are the cases of staphylococcic and pneumococcic tonsillitis, which made up 4 per cent of the 1,022 cases reported in November 1934.

Boese [8] stated that in his experience the thicker the exudate covering the tonsils and the deeper the necrosis the more efficient bismuth has been. Imhof,[9] using this therapy, noted clearing of the purulent exudate from the tonsils after twenty-four hours.

Bismuth apparently has no effect in preventing the formation of peritonsillar abscess. On this point practically all authors have agreed. Buhre [10] made one report to the contrary. Pagano [11] stated that he had encountered 1 case in which the drug prevented the formation of such an abscess and 7 in which it did not and that in the latter cases bismuth made the formation of pus easier, rendering it more fluid. He expressed the opinion that there was quicker healing of the inflammatory process after the opening of the abscess. Those observations were also noted by Falcao and by Silva Guimarães.[12]

If a patient with acute lacunar tonsillitis does not improve within twenty-four hours after the injection of bismuth compounds and does not recover within forty-eight hours, a culture for identification of the causative organism should be made. Experience has shown that

6. Köhler, A.: Die Wismutbehandlung von Anginen in Kindesalter, Kinderärztl. Praxis **8**:194, 1937.

7. Chanine, A. N.: Tratamento das anginas agudas pelo bismuto. (Comunicacão a Sociedade medico-cirurgica de Damasco), Hospital, Rio de Janeiro **14**:279, 1938.

8. Boese, O.: Behandlung der akuten eitrigen Angina mit Wismutsalzen, Deutsche med. Wchnschr. **62**:776, 1936.

9. Imhof, F.: Ein Beitrag zur Wismutbehandlung der Angina, München. med. Wchnschr. **85**:1028, 1938.

10. Buhre, G.: Die Behandlung der Angina mit Wismutinjektionen, Med. Welt **12**:1394, 1938; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **31**:307, 1939.

11. Pagano, A.: Sull'uso del bismuto nel trattamento delle angina acute non specifiche, Valsalva **12**:302, 1936.

12. Silva Guimarães, A.: A bismutoterapia nas anginas agudas (Processo de Aristides Monteiro), Brasil-med. **49**:587, 1935.

in cases such as these the tonsillitis is due to Staphylococcus or Pneumococcus and not to Streptococcus. The latter etiologic agent responds rapidly to injections of bismuth compounds.

This new therapeutic procedure finds its greatest application in children, in whom the disease has an average duration of six to eight days. With bismuth therapy, the duration is shortened to twenty-four hours. A survey of 61 cases has been published by Köhler. Vaz de Mello [18] has presented his observations on the effect of bismuth in gastrointestinal upsets associated with tonsillitis in children. One patient had mucosanguineous diarrhea, tenesmus and elevation of temperature of four days' duration, with thirty-two bowel movements each day. The day after the injection of bismuth compounds was initiated, the temperature became normal, the dysphagia stopped and the bowel movements were reduced to four. The following day the child was well.

Acute tonsillitis in children which does not respond to bismuth within twenty-four hours is usually diagnosed as due to diphtheria, Staphylococcus or Pneumococcus.

The dosage is in accordance with the age of the patient and the solubility of the bismuth preparation employed. I prefer the soluble preparations, because they are more easily absorbed and are quicker in action. For young infants, 1 to 5 mg. of a soluble bismuth preparation and 1 to 2 cg. of an insoluble preparation, according to age, is used. The adult dosage should not exceed 2 cg. of a soluble bismuth compound in twenty-four hours and 5 cg. of an insoluble bismuth compound in each dose. Tonsillitis which is not cured with two doses is not a "bismuth angina," according to Marinho's [14] classification, which divides tonsillitis into "serum tonsillitis" (*angina de soro*), the diphtheria one, and bismuth tonsillitis" (*angina de bismuto*), acute nonspecific tonsillitis.

There are no contraindications except cardiorenal disease or a severe affection of the renal apparatus by an intercurrent disease. In cases in which tonsillitis is the cause of renal impairment, administration of bismuth is indicated to remove the focal element.

The value of the new treatment for acute tonsillitis consists in the reduction of the course of tonsillitis to twenty-four to forty-eight hours instead of six to eight days, in the prevention of complications (Ruttin[15]), in the favorable action on the symptoms of acute tonsillitis and in the improvement of the general and the local condition of the patient.

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13. Vaz de Mello, M.: 130 Fälle akuter Angina bei Kindern durch Wismut geheilt (Methode Aristides Monteiro), Jahrb. f. Kinderh. **145**:203, 1935; A terapeutica bismutica nas repercussões á distancia das anginas agudas, Rev. méd.-cir. do Brasil **45**:21, 1937.

14. Marinho, J.: Une nouvelle classification des angines. Angines à traiter par le sérum antidiphtérique et angines à traiter par le bismuth, Ann. d'oto-laryng., 1935, p. 287.

15. Ruttin, E.: Tonsillogene Sepsis, Monatschr. f. Ohrenh. **72**:379, 1938.

The treatment is economical and simple. A mere prick is enough to guarantee the patient a rapid improvement of the general and the local condition. According to Boese, of Hamburg, Germany, the injection of bismuth compounds is the ideal treatment for acute lacunar tonsillitis, and this statement has been confirmed by many authors.

RECENT FRACTURE OF THE NASAL BASE LINES OF BOTH OUTER NASAL WALLS, WITH DIVERGENT DISPLACEMENT

ORTHOPEDIC PROCEDURES FOR OBTAINING ANATOMIC REDUCTION OF THE OSSEOUS AND CARTILAGINOUS NASAL FRAMEWORK

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ANATOMY OF THE OSSEOUS UPPER HALF OF THE HUMAN NOSE

The outer framework of the upper half of the human nose is made up of an osseous outside casement; i. e., each lateral nasal wall is made up of the flared nasal process of the maxillary bone. Embryologically, the flared nasal process and the maxillary bone are one, and there are no sutures along the nasal base to be separated easily. The osseous roof of the upper half of the human nose is made up of the two nasal bones. The superior edges of these two bones are attached to the frontal bone, while the posterior borders rest on and are attached to the anterior borders of the nasal processes of the two maxillary bones.

The median sutures of the two nasal bones form a definite V, or gable, with each other. The entire septum underlying the upper half of the human nose is osseous. It is made up of the perpendicular plate of the ethmoid and the two plates of the vomer, between which a narrow strip of septal cartilage extends back to the sphenoid.[1] This entire bony septum can be removed or destroyed, and the outer bony casement of the upper half of the nose will still be firm enough to withstand considerable traumatism.

ANATOMY OF THE CARTILAGINOUS LOWER HALF OF THE HUMAN NOSE

The outer framework of the lower half of the human nose, on the contrary, is made up of cartilages, namely, the two upper and the two lower lateral cartilages and the two cartilages of the columella, and soft tissues. It has no osseous structures. All of this cartilaginous structure, especially that portion which extends out into space beyond the maxillary spines, has as its only underlying midsupport the triangular septal cartilage. Thus, when the posterior border of the septal cartilage is dislocated from its anatomic groove in the floor of the nose [2] or when

1. Metzenbaum, M. F.: Submucous Resection for the Correction of Septum Defections with a Description of the Author's Special Instruments, Laryngoscope **21**:86-93 (Feb.) 1911.

2. Metzenbaum, M. F.: Dislocation of the Lower End of the Nasal Septal Cartilage, Arch. Otolaryng. **24**:74-88 (July) 1936.

large portions of the septal cartilage are removed by disease, suppuration or surgical procedure, a loss of the underlying midline supporting structure of the lower half of the nose results, and the nose droops or dips in accordance with the location of the loss of the septal cartilage or of the fractures and displacements.

Fig. 1.—*A*, a patient with recent fracture of the nasal base lines of the outer nasal walls, with divergent displacement, due to severe traumatism at the nasal bridge:

1. The two nasal bones were fractured at their upper third, flattened, depressed and locked between the upper borders of the walls of the nasal processes of the maxillary bones. The resulting increased width at this plane exerted a spreading force, which was transmitted within the nasal processes of the maxillary bones to their nasal base, resulting in divergent fractures of both nasal processes of the maxillary bones along their nasomaxillary or base lines.
2. The perpendicular plate of the ethmoid and the two plates of the vomer, together forming the osseous septum, were fractured, dislocated and depressed.
3. The cartilaginous septum was fractured and dislocated from the anatomic groove in the nasal floor and from the supporting position underneath the soft tissues of the nasal tip.

B, the patient after reconstruction, which was accomplished by reversing the mechanical steps which caused the fractures and displacements of the entire nasal framework:

1. The divergent walls at the fractured base lines of the nasal processes of the maxillary bones were forced toward each other. This procedure narrowed the nasal base and widened the upper borders of the nasal processes of the maxillary bones.

(Legend of Fig. 1 continued on opposite page.)

FREQUENT TYPES OF FRACTURE OF THE NASAL BASE

1. A force directed against the side of the osseous process of the nose will drive the lateral nasal wall toward the septum. If the force is great enough, it will fracture both lateral nasal walls and push the nose off its base, to one side.

Fig. 2.—*A*, side view of the same patient, showing the saddle form of the depressed nasal bridge and the elevation of the nasal tip and retraction of the upper lip. *B* shows how the reduction elevates or raises the nasal bridge, overcomes the "saddle," lowers the nasal tip and the upper lip and reestablishes normal nasal function and normal appearance.

EXPLANATION OF FIGURE 1—Continued

2. From within the nose, the depressed, locked nasal bones were carefully elevated without splintering.
3. The upper borders of the nasal processes of the maxillary bones were compressed to the anatomic width.
4. The two nasal bones were replaced in their anatomic positions on the upper borders of the two nasal processes of the maxillary bones, and the V, or gabled, conformation of the nasal roof was thus restored.
5. The dislocated septal cartilage was reset in the anatomic groove in the floor of the nose. This elevated the depressed nasal tip and restored to it the normal perpendicularity on which normal nasal function depends.

These orthopedic procedures reduced the entire fractured and dislocated framework into the normal anatomic relations and reestablished normal nasal function and appearance.

2. A forceful impact directly over the nasal bridge will result in fractures involving both orbits, both maxillary processes, both zygomatic processes and the antrums. It will split the face along the nasomaxillary base on both sides and through the upper jaw and will drive the nose between the separated maxillary bones, much as a cork is driven partially into a bottle. In an injury of this type there are usually many additional fractures of the facial bones, and in some cases both pterygoid processes are fractured.

THESIS

When a child or an adult meets with not too severe a traumatism directly over the bony nasal bridge, the following conditions result:

1. A depressed locked fracture of the two nasal bones occurs at about their upper third.

2. The two nasal bones no longer form a V, or a gabled roof, to the bridge of the nose but are driven in between, flattened and depressed to the level of the upper borders of the nasal process of the maxillary bones.

3. This flattening and depression of the nasal bones between the upper borders of the walls of the nasal processes of the maxillary bones increases the width at this plane, and a stress is transmitted within the walls of the nasal processes of the maxillary bones to the nasal base, resulting in a divergent or outward spreading fracture of both outer nasal walls along the base lines of both nasal processes of the maxillary bones. A graphic description of the divergent fractured nasal base is that the base of the nose has done the "splits." Fractures and dislocations occur more readily along existing suture lines, but each of these divergent fractures occurs along the base line of the nose, in that part of the maxillary bone which is solid and has a flare and at a place where there is no suture or even an embryologic line of development.

Recent fracture of the nasal base lines of both outer nasal walls with divergent displacement has never been described as a distinct entity. This treatise will deal especially with the mechanical reduction of the divergent fractured nasal base, in order to obtain an anatomic reconstruction of the entire fractured outer and inner nasal framework. My orthopedic methods for obtaining this reduction will be described.

DIAGNOSTIC SIGNS

The following signs are characteristic of fracture of the nasal base with divergent displacement:

1. On inspection, the bridge of the nose is definitely flattened.

2. The base of the nose is spread, or widened, because of the divergent fractures of the nasal processes of the maxillary bones on both sides along the base lines.

3. The osseous upper half of the nose or even the entire nose is brought closer to the face.

4. On palpation, the anterior free sharp borders of the nasal processes of the maxillary bones can be felt distinctly on a level with or slightly higher than the fractured, depressed and locked portions of the nasal bones. This is due to the fact that the lower ends of the fractured nasal bones are driven in between the upper borders of the walls of the nasal processes of the maxillary bones.

5. If a hypodermic needle with a syringe attached is introduced at a right angle to the nasal process of the maxillary bone anywhere along the osseous nasal base on either side, the needle can be forced readily between the maxillary bone and the separated nasal process of the maxillary bone, and it can be seen to enter the nasal cavity between the outer nasal wall and the inferior turbinate. If the hypodermic syringe is filled with mercurochrome, this red solution can be seen flowing in the inferior nasal fossa. After traumatism of the type described in the foregoing sections, these procedures give further proof of a fracture along the naso-maxillary line or the nasal base.

6. Roentgenograms show bony separations. In a fracture of this type the posterior borders of the fractured nasal processes of the maxillary bones are forced outward but are held close to the maxillary bones, so that a roentgen plate which is taken laterally is so far away that the fracture along the base line is seldom revealed. However, fracture of the two nasal bones usually can be demonstrated by roentgenograms.

FRACTURE AND DISLOCATION OF THE OSSEOUS SEPTUM

When the two nasal bones are depressed, the bony septum must give way, for when the nasal roof is lowered, there is no longer sufficient height for the osseous septum to remain perpendicular. The following conditions result:

1. The bony plates of the septum (the perpendicular plate of the ethmoid and the two plates of the vomer) separate along the borders of the narrow strip of the quadrangular septal cartilage lying between them, forming a deep concavity of the osseous septum on one side and a marked convexity on the opposite side (true osseous nasal spur).[1]

2. The two parts of the bony septum may overlap.

3. In very young children the bony parts of the septum may buckle, resulting in a greenstick fracture underneath the two layers of the periosteum.

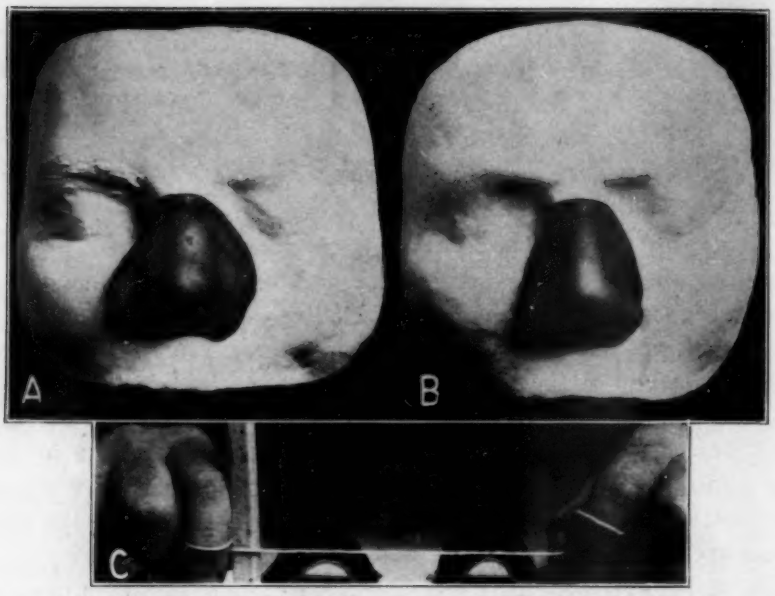

Fig. 3.—Plaster casts were made over the nose and face of the same patient. Dental sheet wax was molded over the nose, to define the nasal outlines. Metal was poured into these wax models, so as to give exact cast, fitting perfectly over the plaster models. *A*, metal cast of the plaster model of the traumatized nose, defining the outlines of the spread nasal base and showing the broad depressed nasal dorsum. *B*, exact metal cast of the plaster model of the reconstructed traumatized nose, defining the narrowed outlines of the reconstructed nasal base and showing how the nose is raised and the dorsum narrowed. *C*, comparison of the heights of the metal casts made over the plaster models of the traumatized nose before and after reconstruction. The measurements were made at the nasal bridges. The cord shows that the depressed nasal bridge was raised ¼ inch (0.5 cm.) by the reconstruction.

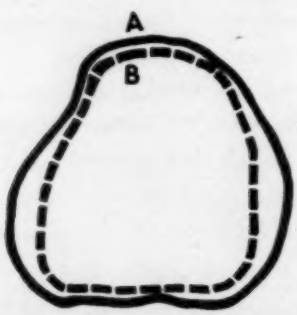

Fig. 4.—Outlines of the metal casts showing: (*A*) the divergent nasal base before reconstruction and (*B*) the narrowed nasal base after reconstruction.

FRACTURE AND DISLOCATION OF THE CARTILAGES AND THE CARTILAGINOUS SEPTUM

After fracture and dislocation of the cartilages and the cartilaginous septum, the cartilaginous elements comprising the lower half of the nose may still remain approximately in their anatomic positions.

More frequently, however, the upper borders of the upper lateral cartilages are torn loose from their attachments to the lower borders of the nasal bones and to the lower borders of the nasal portions of the maxillary bones, but the cartilages usually are held within the peri-

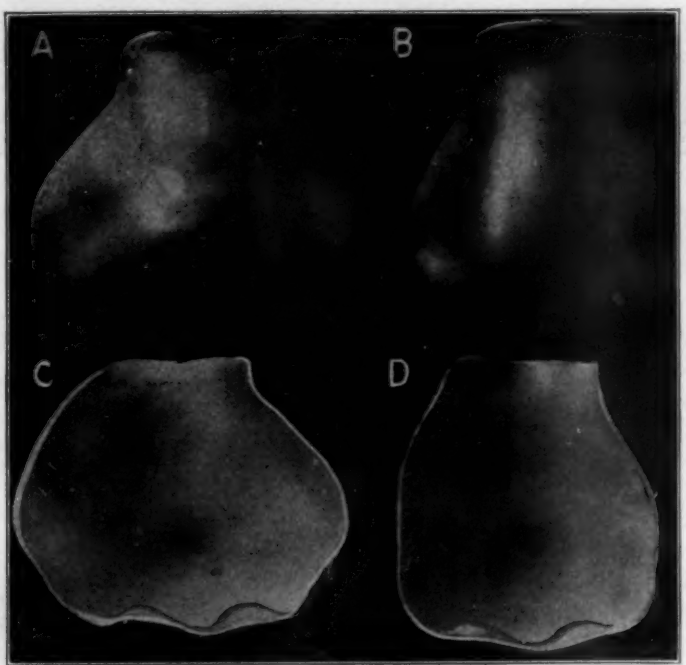

Fig. 5.—*A*, outside view of the metal cast made over the plaster model of the traumatized nose, defining the limits of the spread nasal base and demonstrating the depressed and broad nasal bridge. *B*, outside view of the metal cast made over the plaster model of the reduced traumatized nose, showing how the reconstruction narrowed (1) the nasal base and (2) the dorsum of the nose. *C*, inside view of the metal cast made over the plaster model of a traumatized nose, defining the limits of the spread nasal base. *D*, inside view of the metal cast made over the plaster model of the reduced traumatized nose, showing how the reconstruction narrowed the nasal base.

chondrial coverings of both the outside and the inside linings, so that when the osseous processes are reduced, the displaced cartilages assume their anatomic positions.

The two lower lateral cartilages and that part of each lower lateral cartilage which curves to form the thin columnar cartilage, all of which together make up the freely movable lower cartilaginous process of the nose, are seldom injured.

The superior border of the quadrilateral septal cartilage, which has a simple edge to edge attachment to the inferior border of the perpendicular plates of the ethmoid and the superior edge of the vomer, will be torn loose from part or all of these attachments, but if it is held between the perichondrium of the cartilage and the periosteum of the bony septum, it will be returned to its anatomic position when the fracture is reduced and will be held in that position.

In an injury of this type there is usually a dislocation of part or all of the lower border of the septal cartilage from its anatomic groove in the floor of the nose, as well as a perpendicular or oblique fracture of the extreme lower end of the cartilage where it ends and where it is held between the two maxillary spines. The result of these alterations is that the free border of the cartilage, covered by skin, lies partially across one nostril and, in the opposite nostril, at a higher plane, the fractured cartilage forms an angle of deflection, partially occluding this nostril. This deflection of the cartilage causes an asymmetry of the nostrils, the tip of the nose is lowered and one nostril is flattened and lengthened like the repaired nostril of a patient with cleft palate (harelip). This condition can be recognized by the asymmetry of the nares.[3]

USUAL PROCEDURE FOR REDUCING A RECENT NASAL FRACTURE

The usual method of reducing a recent nasal fracture is as follows:

After the induction of local or of general anesthesia, two straight or curved Kelly hemostats are introduced into the nose for an indefinite distance—sometimes only into the tip of the nose, at other times up to the frontal bones and, unfortunately, sometimes as high as the cribriform plate. The surgeon then elevates the instruments, wiggles the patient's head from side to side and often lifts the head off the operating table. This procedure produces a great deal of intranasal traumatism and may be responsible for the opening of direct pathways to the base of the brain, where before the manipulation the mucous membranes still covering the fine fractures of the nasal roof may have been intact. By this manipulation the fractured lower ends of the locked nasal bones are usually splintered before they can be elevated, but inasmuch as the upper borders of the walls of the maxillary bones are still apart, the fractured and now splintered ends of the nasal bones may fall back between these upper borders of the nasal processes of the maxillary bones or they may rest obliquely to the right or the left of them. They are seldom returned intact to their anatomic positions.

Unless it is recognized and reduced, a divergent displacement leaves the nasal base very broad (negroid in type). Fractures and dislocations

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3. Metzenbaum, M. F.: Asymmetry of Nares: Positive Diagnostic Sign or Entity Establishing Anatomic Displacement of Lower End of Cartilaginous Nasal Septum, Arch. Otolaryng. **16**:690-697 (Nov.) 1932.

of the osseous and cartilaginous septum, unless they are recognized and reduced, leave one or both nares partially or totally occluded, anteriorly, posteriorly or superiorly.

REDUCTION OF RECENT FRACTURE OF THE NASAL BASE LINES WITH DIVERGENT DISPLACEMENT, BY THE AUTHOR'S ORTHOPEDIC PROCEDURE, WITH THE AID OF LOCAL OR COMBINED LOCAL AND GENERAL ANESTHESIA

Both nostrils are packed with a 10 per cent solution of cocaine with epinephrine, to produce intranasal anesthesia and ischemia and to remove

Fig. 6.—A no. 23 short beveled 1⅛ inch (3.4 cm.) hypodermic needle is shown inserted in the region of or into the infraorbital canal, where 0.5 cc. of a 2 per cent solution of procaine hydrochloride with epinephrine hydrochloride in a concentration of 1:50,000 is injected. The needle, without being withdrawn from the tissues, is shifted to the nasomaxillary region, where another 0.5 cc. of anesthetic solution is injected. This will render the nasomaxillary portion of the malar bone anesthetic for two to four hours, making it possible either to reduce a recent fracture of the nasal base or, in an old fracture, to separate by saw or chisel the bone along the entire nasomaxillary base line. This same procedure is repeated on the opposite side.

A no. 23 short beveled 1⅛ inch hypodermic needle is shown inserted in a downward direction just back of the columella through the membranous nasal septum until it contacts the maxillary bone. It will then enter or be in the region of one of the anterior palatine nerve canals, where 0.5 cc. of a 2 per cent solution of procaine hydrochloride containing epinephrine hydrochloride in a concentration of 1:50,000 is injected. Without withdrawing the needle from the tissues, it is then shifted about ⅛ inch (0.8 cm.) to the side, where it will enter or be in the region of the other anterior palatine nerve canal, where another 0.5 cc. of anesthetic solution is injected. This will render anesthetic for two to four hours the two central and two lateral teeth, the middle of the upper jaw and the anterior portion of the nasal floor so that a nasal or an oral operation can be performed on the osseous structures in this region.

as much edema and swelling as possible from the inferior and the middle turbinates and from all the intranasal mucous membrane tissues, so that both nares can be viewed anteriorly into the nasopharynx from the nasal floor to a point above the middle turbinate and so that the intranasal fractures and dislocations of the nasal roof, of the walls of the antrums, of the turbinates and of the osseous and cartilaginous septum can be determined definitely.

Nasal Block Anesthesia (My Method).—With a no. 22 or no. 23 short beveled needle, both infraorbital nerves are injected with a 2 per cent solution of procaine hydrochloride and a 1: 50,000 solution of epinephrine hydrochloride. Through the same needle puncture the nasal base on both sides is infiltrated along the nasomaxillary lines with 1 cc. of procaine hydrochloride. At the base of the columnella a second block anesthesia is made by injecting 1 cc. of procaine hydrochloride into the region of the right and that of the left anterior palatine nerve. It is seldom necessary to make injections into the region of the dorsum of the nose, but injections into this region can be made intranasally, either at the lower borders of the upper lateral cartilages or externally through the skin.

In cases in which general anesthesia is induced, this same block anesthesia is employed, because it lessens the amount of bleeding and the degree of general anesthesia required. Intranasal anesthesia is also induced, with a cocaine-epinephrine pack, so that the septum, the nasal roof, the turbinates and the lateral walls can be seen more readily and their condition determined.

In the reduction of recent fracture of the nasal base lines of the outer nasal walls, with divergent displacement, the important fracture dislocations are those of the divergent or outward spreading nasal walls of the maxillary bones along the nasomaxillary lines or nasal base lines. Before the fractured depressed and locked nasal bones can be elevated and replaced to reform the gabled nasal roof and before the fractured dislocated osseous and cartilaginous septum can be reduced into its anatomic position, these outward spreading nasal walls must be reduced into their anatomic positions.

Orthopedic Procedure.—The operator's thumbs are placed at the base of the nose, on the sides of the nasal processes of the maxillary bones. Then a firm continuous bilateral pressure is exerted against the two divergent lateral nasal walls until they are forced toward each other into their normal anatomic positions. This procedure narrows the nasal base, and at the same time it causes the upper borders of the walls of the fractured nasal processes of the maxillary bones to spread slightly farther apart, so that the depressed locked lower ends of the nasal bones can be unlocked more readily and elevated without splintering. A firm thin flat rubber-covered instrument is placed on the outside of the nose and measured from the nasal tip upward, so as to extend not higher than two thirds of the length of the fractured ends of the nasal bones. The

instrument is then introduced into the nose, along its roof, while the thumb is held at the end of the nose, so that the instrument does not penetrate to a higher level than the measured distance. This is important: The depressed locked lower ends of the nasal bones can be elevated more readily if the elevating instrument does not extend beyond their fractured upper borders. Furthermore, if the elevating instrument is introduced into the nose no higher than the fractured ends of the nasal bones, it is not likely to transmit any force to other intranasal structures

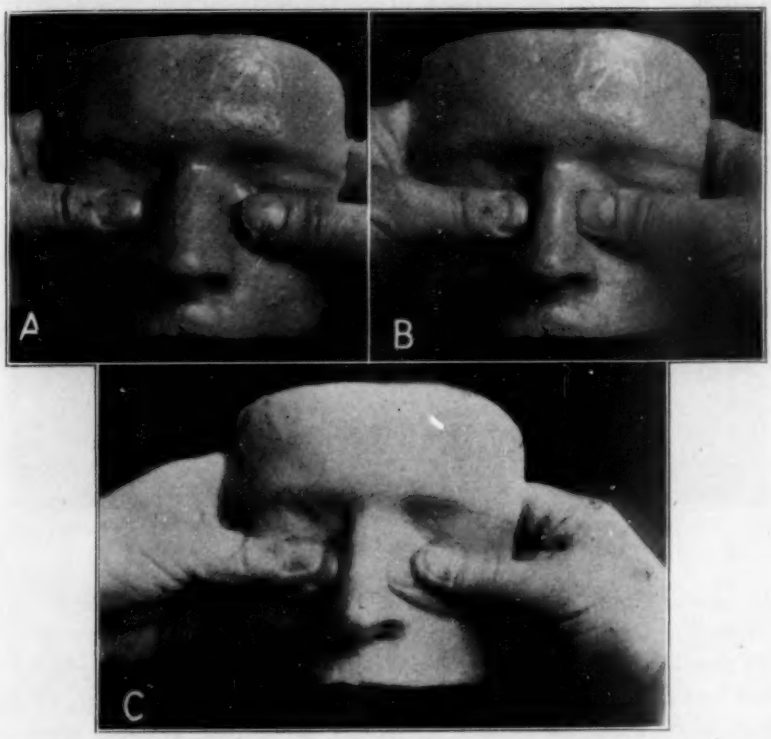

Fig. 7.—*A* and *B*, the exact position of the thumbs in reducing the divergent nasal base. By firm continuous bilateral pressure the divergent lateral nasal walls are forced toward each other into their normal anatomic positions. *C*, Doubly exposed photograph. This gives an idea of the movements of the thumbs necessary to compress the nasal base.

or to the cribriform plate and therefore is not likely to tear any intact mucous membrane which may still be covering a fine fracture of the base of the skull higher up in the nose.

The fractured, depressed and locked parts of the two nasal bones are elevated. Then the upper borders of the nasal processes of the two maxillary bones are slightly compressed toward each other by placing the thumbs along the anterior borders of the nasal processes in a manner

similar to the compression of the nasal base but requiring much less force. The lower borders of the two nasal bones are again placed like a gabled roof in their anatomic positions over the anterior borders of the nasal processes of the maxillary bones.

The narrowing of the nasal base and the elevating of the two depressed, locked nasal bones reestablish the normal perpendicularity, or the normal anatomic height, of the nose, so that the fractured or overlapping portions of the bony septum and the fractured dislocated cartilaginous septum can be made to assume their normal height or their normal perpendicularity in a flat plane. Normal breathing depends on the normal perpendicularity of the lower end of the septum.[2] This is accomplished by adjusting the dislocated fractured portions of the osseous and cartilaginous septum in the midplane by means of a flat rubber-covered instrument or by an Adams forceps, the blades of which are covered with thin rubber tubing. A single Simpson compressed cotton

Fig. 8.—An Adams forceps covered with rubber tubing.

splint is introduced into each nostril. This splint can be made to swell by dropping hydrogen peroxide along the edges, or it will swell in a short time by absorption of the blood serum. A splint of this type forms a true cast of the inside of the nose and exerts a pressure which, though gentle, is sufficient to prevent hematoma or profuse bleeding after the ischemic action of the cocaine and epinephrine on the nasal tissues has worn off.

Strips of adhesive plaster are carefully adjusted to the outside of the nose. They also exert a gentle additional pressure which aids in the prevention of hematoma. Over the adhesive plaster a no. 18 soft copper splint is applied and strapped into place. A U-shaped ice bag is placed over the bridge of the nose, and the patient is placed in bed in a semi-upright position. This position favors seepage into the nasopharynx. On the following day the copper splint and the adhesive plaster are removed, and the Simpson intranasal splints are withdrawn. This is possible without fear that any of the reduced parts of the nose may

become displaced, because the muscles of the face and nose are in pairs and, unlike the muscles of the long bones, have no flexors or extensors to pull the nose to one side. Therefore, a spontaneous redislocation of the nasal framework never occurs.[2]

To afford assurance and to keep the soft tissues close to the bony framework, the nose is again strapped with adhesive plaster for a period of from seven to ten days. The plaster is removed every day or two as the swelling and edema subside.

SUMMARY

I consider the type of recent fracture of the nasal base lines of the outer nasal walls with divergent displacement which has been described here an entity which has not heretofore been classified. When the reduction is carried out by my orthopedic procedure, as described, there result a normal anatomic restoration of the entire osseous and cartilaginous nasal framework and the reestablishment of normal nasal function and normal appearance.

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VASOMOTOR RHINITIS

ITS CAUSATION AND TREATMENT

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From the standpoint of diagnosis and treatment, vasomotor rhinitis may be classified as acute and chronic.

In the first classification, acute, are included all types of rhinitis which are caused by a demonstrable specific inhalant. This category, often referred to as allergic rhinitis, includes hay fever and rose fever. The diagnosis of these types of rhinitis is seldom difficult, because (1) the history of seasonal or periodic incidence is significant and (2) the sensitivity of the patient to a specific allergen is readily demonstrable. Likewise, the treatment of these types of rhinitis is usually gratifying and frequently spectacular in effectiveness.

The second classification may be designated as chronic vasomotor rhinitis. In contrast to the patient of the first group, patients with rhinitis of this type experience no periodic remittance of their symptoms but are continually troubled in varying degrees. Moreover, although this condition has been referred to as a type of allergic rhinitis, more often than not one is unable to demonstrate hypersensitiveness of the patient to any specific irritating allergen.

The classification is complicated by the fact that cases are often encountered in which the two types of vasomotor rhinitis coexist—the acute specific seasonal type and the chronic, perennial apparently nonspecific type.

This paper will be limited to a consideration of chronic vasomotor rhinitis. It is my belief that this is a general disease with marked local manifestations rather than a purely local nasal condition. However, the nasal symptoms are usually so pronounced that other manifestations of the disease are overshadowed, and it is from a rhinologist that the patient generally seeks relief.

The usual complaints noted subjectively are sneezing, interference with breathing, caused by blockage of the nasal passages, and a profuse watery nasal discharge. These symptoms are often accompanied with headache and an irritative cough. Usually several or all of the symptoms are present in varying degrees, and the patient often appears to be in marked distress.

On examination one finds that the mucous membrane is pale and boggy, apparently water logged, and is covered by a serous discharge.

Frequently the turbinates are so swollen that they touch the septum, and occasionally polyps are present. The sinal ostiums are often occluded, a condition which accounts for the headaches.

This picture is so typical and subject to so little variation that the diagnosis of chronic vasomotor rhinitis is almost self evident. It is when the question of treatment arises that the problem becomes difficult and in many cases baffling. Many types of intranasal therapy have been advocated, all effective to a greater or less extent. In a later portion of this paper, the most popular types of local treatment will be outlined and their relative merits discussed. But it is my conviction that even the best of these treatments are only palliative and that to secure anything more than temporary amelioration of symptoms the patient must be studied and treated from a general point of view.

Earlier in the paper I mentioned that I believe vasomotor rhinitis to be a local manifestation of some general imbalance or maladjustment. I feel that one's best chance of clearing up the nasal condition is by discovering and treating the disease at its source.

If one considers in detail the anatomy and physiology of the normal nasal mucosa, one may arrive at the answer to the question of the remote cause of the changes of the mucosa in vasomotor rhinitis.

The outstanding characteristic of the tunica propria, or stroma, of the respiratory portion of the nasal mucosa is that it contains an abundance of mucus-secreting glands, serum-secreting glands and mixed glands and an extremely rich blood supply. This is especially true of the portion covering the inferior and middle turbinate bones. These blood vessels and glands in the stroma are innervated by the autonomic nervous system through two separate and antagonistic sets of fibers, the vasoconstrictors and the vasodilators.

The vasoconstrictor fibers, also called the sympathetic fibers, or pressors, are derived chiefly from the cervical sympathetic and to a lesser extent from the vidian nerve. The vasodilator fibers, also called the parasympathetic fibers, or depressors, comes from the vidian nerve. The impulses from both sets of fibers are transmitted to the nasal mucosa by way of the sphenopalatine ganglion.

In the normal state the two sets of nerve fibers maintain a physiologic antagonism. The predominance of one group over the other, either by increased stimulation of one or reduced stimulation of the other, results in vasomotor changes in the nose. In vasomotor rhinitis there are probably increased stimulation of the parasympathetic fibers, giving greater vasodilatation, and lessening of the impulses from the sympathetic fibers, causing weakened vasoconstriction.

It is not clearly understood whether it is this vasodilatation or, as some writers believe, increased permeability of the capillaries or a combination of both that brings about the characteristic edema of the nasal

mucosa. This edema is seen as a marked outpouring of extracellular or interstitial fluid in the spaces between the connective tissue fibers of the tunica propria, or stroma (subepithelial layer), of the mucous membrane. Another element in the swelling that is apparent is the change which occurs in the glands of this layer. The gland ducts become obstructed and the glands fill up with secretion. Eventually they degenerate into cysts.

From the standpoint of therapy it is important to point out the chemical composition of the interstitial fluid mentioned. This fluid, as well as that inside the cells, is derived from the blood serum, but it differs markedly from the intracellular fluid in that it contains large amounts of sodium as a base, whereas the intracellular fluid contains no sodium but has potassium as the entire base.

The presence of edema has, therefore, some connnection with the amount of sodium in the body fluids. And in passing it should be mentioned that the agent which maintains control over the sodium in the body seems to be of hormonal nature, probably originating in the adrenal cortex.

With these physiologic and pathologic facts in mind it becomes apparent that a therapeutic program should include a consideration of the autonomic nervous system and its association with the endocrine glands.

It is more than a coincidence that patients suffering from vasomotor rhinitis often show symptoms of an imbalance of the autonomic and endocrine systems. These are often overlooked because of the predominance of the nasal symptoms, but on close questioning and observation one frequently finds that one is dealing with emotionally unstable, sensitive persons from the higher walks of life. Patients with vasomotor rhinitis seen at clinics often trace the onset of their illness to the time when they experienced financial reverses. Persons of worrisome and tense nature are made more irritable by the nasal obstruction and headaches, and so a vicious cycle is initiated. Patients with an autonomic imbalance are often observed to suffer from endocrine disorders of greater or less degree. In this group are a surprisingly large number with hyperthyroidism and ovarian dysfunction. Maladjustments of sex are frequently brought to light. In short, the majority of the cases of apparently simple rhinitis present on investigation a picture of generalized glandular and vasomotor malfunction.

Granting, then, that patients with vasomotor rhinitis must be treated from the general as well as from the local point of view, one runs into difficulties when it comes to therapy, as there is no known specific which will control the involuntary nervous system. Therefore, treatment must be planned along empiric lines.

GENERAL TREATMENT

Psychotherapy.—Psychotherapy, including autosuggestion, although taxing the patience and resources of the physician, should be tried, as it often works wonders for emotionally unstable worrisome patients. It is with persons such as these that cures are obtained through non-medical cults, such as Christian science.

Glandular Therapy.—If some glandular dysfunction is suspected, the aid of an endocrinologist will often bring gratifying results.

Drug Therapy.—Some drugs are useful as adjuvants in the treatment:

(*a*) Sedatives, such as phenobarbital, are especially indicated for nervous, high strung patients.

(*b*) Atropine, which, being a vasoconstrictor, tends to lessen the watery nasal secretion, may be combined with phenobarbital. However, atropine is not indicated for prolonged use, because of its general drying effect, the vasodilatation of the eyes which follows its use and other undesirable side effects.

(*c*) Ephedrine also may be used, either alone or in combination with phenobarbital. Ephedrine is not suitable for continued administration.

(*d*) Calcium in the form of calcium gluconate when administered intravenously often has a marked beneficial effect both locally on the nose (by causing vasoconstriction) and generally on the autonomic nervous system.

(*e*) Potassium is an important drug in the rhinologist's armamentarium. If one bears in mind the fact brought out in an earlier section of this report that the interstitial fluid (edema) in the tunica propria of the nasal mucosa is rich in sodium and poor in potassium, the rationale for the use of potassium becomes apparent. However, instead of using the drug per se, it is preferable to administer it by means of potassium-rich foods, in which form, I believe, it is better absorbed. The problem of diet will be discussed fully under that heading.

Diet.—It is theoretically conceivable that an increase of sodium in the body is due to a pouring out of medullary hormone (which is the regulator of sodium metabolism) as a consequence of some dysfunction of the autonomic nervous system. At any rate, it is known that when sodium is increased there is an increase of interstitial fluid, or edema of the nasal mucosa, and, conversely, that a decrease in sodium will cause a lessening of the edematous condition. This is the rationale for the dietary therapy, which consists in providing a salt-free, antiretention, high protein diet.

A diet is chosen which is rich in potassium-containing foods and low in sodium. Moreover, as most of the sodium enters the body in the form of table salt, this is withdrawn and a seasoning substituted which, although tasting like salt, contains no sodium. Occasionally it is advisable to add potassium to the diet in the form of potassium chloride or potassium iodide. (The latter is especially indicated if there is an asthmatic complication.)

Another method of increasing the potassium in the interstitial fluid is by giving a high acid (high protein) diet, for it has been shown experimentally that acidifying of the blood serum tends to dissociate the potassium ions from their combination with the proteins and lipoids of the serum and allows them to diffuse into the interstitial fluid.

This is the rationale for the high protein salt-free antiretention diet I prescribe.

As an adjuvant to the diet, a polyvitamin capsule is prescribed, in order to prevent avitaminosis.

Patients usually lose some weight under this regimen (partly on account of the lessening of edema of the tissues). It is therefore advisable to keep a weight record for each patient. If the loss of weight is considerable, the diet must be readjusted.

LOCAL NASAL TREATMENT

While a patient is being studied in the hope of finding clues to some autonomic dysfunction, it is advisable to give him symptomatic relief by local treatment to the nose. Of the many varying types of therapy that have been advocated, several have considerable merit and will be outlined in some detail.

Therapeutists agree that before beginning treatment directed toward decreasing the bogginess of the nasal mucosa and restoring it to a healthy state one must correct any concommitant contributing pathologic condition which is present in the nose. If polyps are present, they should be removed, either by means of a snare or by electrocoagulation. If there is a deflection of the nasal septum great enough to cause serious obstruction to the nasal passages, a submucous resection of the septum should be done. In passing, however, it should be emphasized that conservatism is imperative in treating vasomotor rhinitis, and radical operative procedures should be frowned on unless there is a definite serious indication for their use.

As for office procedures directed to giving palliative relief, the following are to be recommended:

Zinc Ionization.—By zinc ionization a pale, boggy membrane is changed to a redder, dry membrane. This change is accompanied with great improvement of the subjective symptoms. The application of this

therapy is relatively simple. Any galvanic machine may be used, but the smoother the current the better the result. This fact seems to indicate that direct current and battery sets are superior to machines which operate on alternating current.

As a preliminary, the patient is given a sedative, such as phenobarbital or morphine sulfate combined with atropine. The ionization is carried out as follows: First, the nasal mucosa is shrunk with a 5 per cent solution of cocaine. Then the nose is well packed with strips of cotton soaked in a 1 per cent solution of zinc sulfate. Care should be taken to surround the inferior turbinate and also to avoid the olfactory area. Into the middle of this packing a pure zinc electrode is inserted. The electrode is attached to the positive pole, and the negative pole is the indifferent one. A current of from 10 to 15 milliamperes is applied for about fifteen minutes, although the time is subject to some variation. Only one side is treated at a sitting. Immediately after the treatment, a grayish white covering is seen on the nasal mucosa. Within a few days it is replaced by a thick membrane, which sloughs away, leaving a membrane which is deep red and shrunken.

The improvement following a single treatment may last from several months to a year. Ionization may be repeated with no visible deleterious effects on the membranes. In my hands the results obtained with zinc sulfate solution have been superior to those from zinc sulfate-acacia jelly or copper sulfate solution. (This treatment is of little use in any type of acute vasomotor rhinitis, such as seasonal hay fever.)

Phenolization.—The application of pure phenol to the nasal mucosa gives end results similar to those obtained from ionization. The respiratory portion of the mucosa is swabbed with phenol, care being taken to have no excess fluid drip back into the nasopharynx. The effect is not so lasting as that of ionization; otherwise, phenolization compares with that therapy. Besides, it has the advantage of not requiring any special apparatus.

Electrocoagulation.—In some cases submucous coagulation of the turbinates is helpful. A single-acting or double-acting electrode is inserted into the submucous membrane for the entire length of the inferior turbinate and the current is applied. This results in submucous shrinking of the mucosa. The middle turbinate may also be treated in this fashion. Coagulation increases the airway, making the patient more comfortable. The benefits obtained are of variable duration but often last several months.

Irradiation.—In cases in which polyps have been found and removed, the application of radium to the mucosa is frequently advisable to prevent their recurrence. At the same time it causes marked vasoconstriction of the nasal mucosa, resulting in fibrosis, with diminution of

the bogginess and secretion. The average patient may receive 50 mg. of radium to each side for two hours, making a total dose of 200 milligram hours. At the end of a week the same dose may be repeated or a smaller amount may be given. Results obtained with radium are usually good; however, bearing in mind the potency of radium, one should select with care the patients who are to receive this treatment.

Miscellaneous.—Among the various other treatments recommended is that of injecting alcohol into the sphenopalatine ganglion. If one bears in mind the innervation of the nasal mucosa, it is understandable that this treatment will afford relief. However, the injection of alcohol into this ganglion is painful and not without danger and so is not to be recommended as a routine procedure. The injection of alcohol, the injection of sodium morrhuate and the injection of a 10 per cent solution of phenol into the inferior turbinates have also had their advocates. In my experience none of these methods gives results as desirable as those obtained by zinc ionization, the application of phenol or submucous electrocoagulation.

SUMMARY

Patients with vasomotor rhinitis must be divided into two great groups, one composed of those whose symptoms are caused by a specific inhalant, an allergen, the removal of which allays the symptoms, and the other of those in whom an allergy cannot be observed and in whom the symptoms are perennially present, although in varying degrees. The treatment of chronic vasomotor rhinitis should be fundamentally that of the autonomic nervous system, since the nasal symptoms are only a part of the disease. While searching for the basic cause and eradicating general contributory factors, one must resort to various indirect approaches and, in addition, afford relief to the patient by local intranasal therapy.

151 Central Park West.

ULTRAHIGH FREQUENCY DIATHERMY IN OTO-LARYNGOLOGY, WITH ESPECIAL REFERENCE TO DOSAGE MEASUREMENT

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Modern electric science had its beginning in the year 1600, when William Gilbert (1540-1603) published his epoch-making work, "De Magnete." He discovered that an attractive force could be produced by friction of glass, wax, sulfur, amber and other substances and called this force electricity from the Greek word for amber.

In 1730, Stephen Gray of London first insulated and electrified a human subject, and four years later, the Abbé Nollet first elicited a spark from such a subject. As early as 1746, the latter was treating persons for paralysis with insulation, sparks and shocks.

In 1746 Musschenbrock of Leyden constructed the Leyden jar, and in 1748, Jallabert of Geneva used it to treat a man who had had a paralyzed arm for fifteen years and brought about a cure. Benjamin Franklin at Philadelphia began to treat persons for paralysis with static electricity in 1752, thus starting a widespread medical vogue in this country, reflected in the tremendous literature on the subject. Later, galvanism and faradism were introduced, and for many years these eclipsed static electricity in popularity.

In 1839, Joseph Henry of New Jersey demonstrated that the discharges of the Leyden jar were oscillatory, and this opened the door for the development of high frequency electrotherapeutics. His discovery was amply confirmed in 1847 by Helmholtz and in 1853 by Sir William Thomson (Lord Kelvin).

Holtz in 1865, invented his static machine, which created a new interest in this form of electricity. William J. Morton of New York spent some time in 1880 with Charcot in Paris and became intensely interested in the latter's use of the Holtz machine in the treatment of nervous diseases. On his return to New York he brought along a double Holtz machine, to which he added a pair of Leyden jars. He joined the discharging rods of the machine to the inner coatings of the jars and connected the sponge-covered electrodes held by the patient to the outer coatings. He noted that as soon as the machine was started, there was a soft, agreeable sensation, with no shock or muscular contraction. In

1881, he reported the use of this current in the treatment of various types of nervous diseases, thus being the first to use high frequency electrotherapy.

Morton continued his studies of static induced current and on Dec. 2, 1890 (published 1891) read a paper on its physiologic characteristics. He stated further that frequencies of 100 megacycles to 20,000 megacycles were possible. Sir Oliver Lodge and Heinrich Hertz had, in the meantime, been studying the physical properties of high frequency oscillations and had suggested various means of improving the machines for their production. In addition Hertz (1892) learned that vibratory waves pass from a generator to a distant receiver without tangible connections and thus laid the foundation for radio communication. Since that time, these waves have often been referred to as "Hertzian waves."

In 1891, d'Arsonval of Paris replaced the static machine with commercial alternating current through a Ruhmkorff coil as the source of power, and also placed a coil of fifteen or twenty turns of large wire across the electrode leads. In this way he obtained a current different from any previously used. He found that currents of more than 10 kilocycles could be passed through the body without pain or muscular contraction, that heat was produced and that respiratory activity was augmented.

Also in 1891, Nikola Tesla surrounded the d'Arsonval coil with a secondary coil of many turns and found that the effects were much increased.

In 1892 and 1893, d'Arsonval studied the physiologic effects further, and noted that the passage of high frequency current through the body caused a fall in blood pressure. The heat produced caused the skin to become red and covered with perspiration and resulted in more rapid breathing with a greater absorption of oxygen and a greater elimination of carbon dioxide. When the current passing through the extremity of a rabbit was strong enough, gangrene resulted. He also noted that the current was analgesic.

In 1892, Rivière of Paris in treating a patient with a d'Arsonval machine repeatedly touched a chronic ulcer on the patient's hand with the spark and noted that the lesion underwent rapid healing. Thus it appears that Rivière was the first to use diathermy by fulguration.

Later developments by De Forest were a factor in preparing the way for modern diathermy.

TYPES OF EQUIPMENT

Before proceeding further, it is desirable to classify the different types of diathermic equipment in use at present according to the frequency or wavelength:

1. Conventional, low frequency or long wave diathermy (300 kilocycles to 3 megacycles or 1000 meters to 100 meters; a kilocycle is 1000

cycles per second, and a megacycle is 1,000,000 cycles per second). Conventional diathermy is usually applied by electrodes insulated from direct current voltages and placed in direct contact with the skin. Heating results largely from conduction currents.

2. High frequency or short wave diathermy (3 to 30 megacycles or 100 to 10 meters). This may be applied by pads or cuffs insulated by means of some solid dielectric, the heating being effected by the electric field. In addition, it may be applied by a coil; then the heating is presumably due mainly to the magnetic field.

3. Ultrahigh frequency or ultrashort wave diathermy (30 megacycles to 300 megacycles, or 10 to 1 meter). Satisfactory application of this type of diathermy may be had by means of air-spaced electrodes, the heat being principally due to dielectric losses in the tissues. The effect is similar to the heating of a dielectric material located between the plates of a radio condenser.

4. Waves of more than 300 megacycles in frequency (1 meter or less) are commonly referred to as microwaves. As yet, little medical application has been made of these frequencies.

FIRST STUDIES OF ULTRAHIGH FREQUENCY DIATHERMY

For many years all diathermic equipment was of the conventional or low frequency type. High and ultrahigh frequency diathermy have come into use only relatively recently.

In 1924, Gosset, Gutmann, Lakhovshy and Magrou, working with plant tumors caused by inoculation of Bacterium tumefaciens, noted that these tumors could be eradicated by ultrahigh frequency irradiation. Their apparatus produced waves of 150 megacycles (2 meters), and they irradiated the plants over periods varying from six to thirty-three hours, presumably by placing them in a condenser field. The tumors increased in size for several days, but after about two weeks began to undergo necrosis, usually complete in another two weeks. Healing of the plants followed.

Carrying forward this work on animals, Schereschewsky in 1926 found that mice placed in a condenser field showed agitation; their ears, tails and paws turned bright pink and later cyanotic; there were salivation, sweating and increased nasal secretion; still later there were convulsions with dyspnea, and finally the mice died. The postmortem temperatures ranged from 42 C. (108 F.) to 44 C. (111 F.), indicating that the symptoms were due to heat retention. These effects were most marked with frequencies between 18 and 66 megacycles (16.5 and 4.5 meters).

Esau also published a paper in 1926 on the technical aspects of ultrahigh frequency radiation, using waves of 60 to 300 megacycles. No physiologic data, however, were reported.

In 1928, Schereschewsky reported experiments with transplantable sarcomas from fowl and mice. He found that frequencies of 66 to 68 megacycles would produce complete recession of the tumor and consequent recovery of the tumor-bearing animal. Only 5.5 per cent of the diseased mice died of tumor; those that recovered were, however, more susceptible to bacterial infections than the controls. When the treatment was too severe, death followed. Although this work augured well, Whitney in 1932 reported an unsatisfactory experience. Haas and Lob (1934) and Baumeyer (1938) stated that ultrahigh frequency radiation often caused malignant tumors to increase but not to decrease in size, and they concluded that this type of therapy was not suited to tumors in man.

In 1928, Whitney noted that men working in the field of an ultrahigh frequency radio transmitter experienced a sensation of warmth, even though the surrounding temperature was cold. Hosmer (1928) in further study of this phenomenon found that the temperature of the thorax in rats could be raised as high as 115 F. Whitney found in 1928 that fruit flies could be killed by 1 kilowatt second of radio energy and that dormant refrigerated insects could be made to fly about in a condenser field in an air temperature of 0 C. (32 F.).

Schliephake, in 1928 and 1929, published several reports of studies on the biologic effects of ultrahigh frequency radiation in animals, which confirmed the findings of previous investigators. He noted, too, that if the brain of an animal is heated too long, a fatal disturbance of the temperature-regulating mechanism may result. In 1930, he published the first report of treatment of human beings. Probably the first case of a purulent process treated thus was a furuncle on the nose of Schliephake himself which, he stated, disappeared in twenty-four hours after one treatment. He also reported favorable results in the treatment of other surface infections, such as boils and carbuncles, of certain types of arthritis, and of tonsillitis and sinusitis.

In 1930 and 1931, Carpenter and Boak studied the effect of ultrahigh frequency diathermy on syphilis in rabbits and, finding them improved, extended the work to human beings. Hinsie and Carpenter (1931) noted that their patients tolerated the treatment well and that 11 of 17 patients with dementia paralytica were so improved as to be able to leave the hospital. Studies of the blood of these patients by Hinsie and Blalock (1931) indicated a rise in the leukocyte count, which often started within fifteen minutes after treatment was begun. The polymorphonuclears, especially the nonfilamentous forms, were increased at the expense of the lymphocytes. Knudson and Schaible (1931) found other changes,

such as loss of weight, decrease in blood volume, concentration of blood, increase in nonprotein nitrogen and blood sugar, all of which returned to normal in about twenty-four hours. Rausch (1934) noted similar effects. Jacobsen and Hosoi (1931) concluded that the alterations produced differed little from the effects of fever from other causes.

SPECIFIC EFFECTS

Schereschewsky suggested that these currents might have a specific quality of attacking certain cells more than others and that this might be of value in treatment. Schliephake (1933) went further and stated his belief that every tissue was assigned a certain wavelength at which it was particularly susceptible to heating, although he was not able to demonstrate it. Rajewsky (1938) even thought that this selectivity might be of use in diagnosis. However, Christie and Loomis (1929), Mortimer and Osborne (1935), Curtis, Dickens and Evans (1936) and Hasche (1938) found no specific effects other than heat. The Council on Physical Therapy of the American Medical Association stated in 1939 that "the consensus seems to be that no physiologic effects other than those attributable to heat have been substantiated." This fairly well disposes of the so-called athermic treatment as anything except an ultraconservative form of therapy.

However, Cumberbatch (1937) pointed out that, in general, waves of 60 megacycles (5 meters) to 100 megacycles (3 meters) are best adapted to the treatment of small regions of the body, such as the head.

In connection with specific heating effects of certain regions or organs at particular frequencies, there may be slight tendencies of this nature. However, due to the shunting effects of adjacent regions and the relatively high electric resistance of any path in tissue, the frequency response of any tissue region to high frequencies must be extremely broad. In other words, nothing even approaching a sharply tuned radio circuit exists in tissue. Thus, the heating of any given region by waves of 10 megacycles will be much the same as by waves of 7 or 15 megacycles.

However, with large changes in frequency, such as several hundred per cent, there may be perceptible changes in the heat distribution in the tissues under treatment. For example, Paetzold (1938) found that waves of 300 megacycles (1 meter) heated fats only one third as much as waves of 27 megacycles (11 meters.) Thus, in observing this differential heating effect, Paetzold employed a change in frequency of 1,100 per cent.

A selective action on bacteria in vitro was reported by Haase and Schliephake (1931), but Lentze (1932), Szymanowski and Hicks (1932), Hasche and Leunig (1934), Whitney and Page (1935) and others found no inhibitory or destructive effect.

CLINICAL OBSERVATIONS

The heat generated in the mouth, nose and sinuses is quickly dissipated by the blood. Tebbutt (1935 and 1937) noted an actual decrease of 1 to 2 F. in the antrum following the administration of ultrahigh frequency radiation; he attributed this to evaporation of the increased nasal secretions concomitant with the treatment. Hollender (1939) measured the temperature of the antrum through a window in 20 patients following treatment and noted elevation in some, fall in others and no change in a third group. Laszlo (1939) noted similar results and, in addition, a drop in blood pressure of from 5 to 40 mm., from which there was a quick return to normal. It will be recalled that d'Arsonval made a similar observation in 1893. It would appear that these findings indicate a varying action on the sympathetic-parasympathetic vasomotor system. The beneficial results of treatment cannot be ascribed to the heating alone, but, as Hollender (1939) pointed out, to the resultant hyperemia and hyperlymphia. The sensation of warmth is, however, pleasant and slightly analgesic, so that many patients nearly go to sleep during treatment.

The most acceptable fields for ultrahigh frequency therapy in otolaryngology are acute and subacute infections of the sinuses, cellular tissues of the face and neck, cervical lymph glands and tonsils and nonsuppurative inflammations of the eustachian tube and ear. Our experience coincides in the main with that of Laszlo (1937, 1939), Leichner and Schmidt (1937), Cumberbatch (1937), Egan (1939), Brugsch and Pratt (1939) and Hollender (1939). We agree with the last that the treatment is no panacea but should be used along with other measures. So many variable factors are involved that it is difficult to appraise results, but when these are compared with the results obtained prior to the advent of the modality, there can be no question that most patients recover more rapidly with the added radiation.

DANGERS AND CONTRAINDICATIONS

The early reports of Schliephake (1932) and Liebesny (1935) seem too enthusiastic to most writers. Kowarschik (1936) sounded a note of warning when he stated that every severe infection or cellulitis should be treated under the supervision of an experienced surgeon, who can decide when conservative therapy should be abandoned and operation performed.

There are a few side reactions which require watching. Rausch (1934), Laszlo (1937, 1939) and others have noted a fall in the blood pressure; there is usually a return to normal after a short time, but Schliephake (1935) found the condition unresponsive to epinephrine. Consequently, faintness, dizziness or nausea indicates immediate termination of the treatment.

In acute infections pain may be increased owing to the added hyperemia and hyperlymphia. Our policy has been to delay using radiation until the hyperacute inflammation has subsided.

Brugsch and Pratt (1939) stated that impaired sensation, occlusion of peripheral arteries or veins, advanced diabetes and arteriosclerosis with marked hypertension constitute definite contraindications to this form of treatment. In general, we believe that patients over 60 years of age should be treated conservatively and watched carefully.

MEASUREMENT OF DOSAGE

Until recently, diathermic machines have had no provision for measuring dosage. Without such measurement empiricism ruled, and clinical reports have been difficult to interpret owing to lack of comparability of factors. Heat sensation has been the only method of grading dosage, but as Kowarschik (1939) pointed out, this is no criterion, since much of the heating occurs in deep tissue, and there are no temperature receptors below the skin level. The result is that burns have developed, as reported by Kovacs (1935), Kling and Berg (1935), Krusen (1935), Schmidt (1935), Coulter (1935), Egan (1939) and others. Undoubtedly many have occurred which have not been reported. It is apparent that such a situation could easily lead to malpractice involvements and possibly to the discrediting of all ultrahigh frequency diathermy. The necessity for control of dosage is obvious.

In most diathermic equipment the only indicator in the circuit passing through the patient is an ammeter in series with one of the electrodes. This meter gives no indication of the actual power absorbed by the patient but serves merely as a tuning indicator.

The patient receives only part of the radio frequency power output of a diathermic machine. The remainder of the output is either radiated or dissipated as heat in the conductors or dielectrics of the circuits. Thus the total power output of the generator is divided between circuit losses, radiation losses and power effective on the patients. The latter portion is the only one in which the therapeutist is interested and which he would like to measure.

As previously mentioned, the physiologic effects of diathermy appear to be largely those of heat. Heat is a form of energy. Power, on the other hand, expresses the time rate of expenditure of energy. Thus, once the average power and the exposure time are known, the total energy or heat is known. Accordingly, heat = energy = power $\times$ time. In the case of diathermic treatment the power absorbed by a patient may be designated as the dose rate. The energy absorbed is then the average dose rate times the length of time of treatment. This may be called the dose.

The dose rate may be expressed either in watts or in calories per minute (1 watt equals 14.3 Gm. of calories per minute). The dose then is expressed either in watt minutes or in calories. For example, a patient receiving power at an average dose rate of 20 watts for fifteen minutes receives a total dose of 300 watt minutes or 4,290 calories. In specifying a dose, therefore, both the dose rate and the time should be given.

The first practical method for measuring the dose rate was published by Mittelmann in 1937. Other papers by Mittelmann (1938 and 1940) discussed the problem further.

An ultrahigh frequency diathermic machine incorporating means for measuring the power absorbed by the patient was completed by us early in 1940. The power measurement method follows the one described by Mittelmann and has been found to be entirely satisfactory in practice. Our equipment is shown in the photograph. A description and complete wiring diagram together with an analysis of the power measurement method has already been published (Kraus and Teed, 1940). However, we shall give a brief description here and mention some of the unique features of the method.

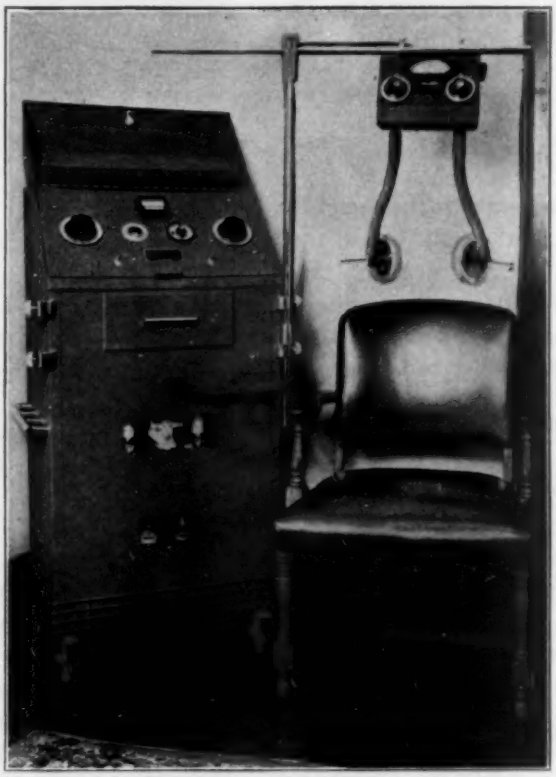
The castor-equipped larger unit houses the radio frequency generator, power supplies, switching and control equipment. All controls are grouped on the sloping panel at the top. A hinged cover, which is open in the photograph can be lowered over this panel and locked when the machine is not in use. Frequency and power controls are provided, as well as a time switch for automatically shutting off the machine at the end of the desired treatment period. The machine is capable of generating radio frequencies between 33 and 50 megacycles, although a frequency of about 42 megacycles (7 meters) is usually employed.

The smaller unit or dosimeter is supported from the larger unit by adjustable brackets as shown. This unit in turn carries the applicator electrodes by means of insulated gooseneck supports. The electrodes are copper disks 9 cm. in diameter and are encased in a hollow plastic housing with a 2 cm. air space between electrode and plastic case. The dosimeter is coupled to the larger unit by means of a flexible low loss transmission line.

In treating sinusitis, the electrode housings are placed laterally against the face, with the anterior and inferior portions of the plates closer together than the posterosuperior. This serves to concentrate the radiation in the sinus area and away from the brain. It is not advisable to place the electrode housing against the tip of the nose, since, as Kling and Berg (1935) and de Cholnoky (1937) pointed out, the nose localizes the heating and may be burned.

The procedure in using the machine is as follows: After adjusting the electrodes to the patient, the entire dosimeter unit is swung away

from the patient on a swivel incorporated in the bracket. The machine is then turned on, and the electrode circuit resonated by means of the tuning control on the dosimeter unit. The indicating meter is set to a predetermined value, and the electrodes are swung back into position on the patient. The electrode circuit is reresonated, and the new value of the indicating meter observed. The calibrating control is then set at the value observed. This procedure automatically calibrates the dosimeter



Ultrahigh frequency machine. The larger unit houses the generator, power supply and controls. The bracket-mounted smaller unit contains the dosimeter. From the large indicator on this, power absorbed by the patient may be read directly.

for the individual patient. Following this adjustment, the indicating meter then reads directly the dose rate or power input to the patient. This dose rate may then be adjusted to any desired value by means of the power control on the larger unit.

The over-all absolute accuracy of the machine appears to be within about 15 per cent, thus comparing favorably with determinations used at present in roentgen therapy.

RADIATION LOSSES

Not only does the power radiated represent a loss, but it also constitutes a source of serious radio interference.

In ultrahigh frequency machines having relatively long, widely spaced leads to connect to the applicators, much power may be radiated from the leads themselves. With such leads, the system may approach the dimensions of a "flat-top beam" antenna, which consists of two closely-spaced out-of-phase dipoles. It has been shown that even when the radiating efficiency of such an antenna is reduced by the presence of large values of loss resistance, a considerable fraction of the power input to the antenna may, nevertheless, be radiated. As an antenna, the efficiency may be low, but as a device for transferring power to a patient, it represents excessive radiation (Kraus and Teed, 1940).

Our machine has been designed throughout with a view to reducing radio interference to a minimum. By using closely spaced short leads from the dosimeter to the applicator electrodes, the radiation from the circuit passing through the patient is greatly reduced. At a frequency of 42 megacycles (7 meters), the leads are less than 0.05 wavelength long and about 0.03 wavelength apart. Both the larger and the smaller units are all of metal construction, thus providing shielding and reducing radiation from the circuits they contain. The flexible transmission line connecting the units is of the balanced two wire type, from which radiation is negligible. It has been found that as a result of this design the radiation on the fundamental frequency (normally about 42 megacycles) is greatly reduced.

By using full wave rectification and adequate filtering of the anode voltage supply and by locating a radio frequency filter in the power line where it enters the main unit, radio interference on frequencies other than that of the fundamental has been eliminated. Thus, it is possible to operate a conventional "all band" receiver immediately adjacent to the machine with no interference.

The discussions of Coulter, Bierman and Hansson (1939), Krusen (1939) and others seem to argue that the radio services should overlook interference from diathermic equipment, since the latter is being used in the cause of alleviating human suffering. However, we feel that proper design can reduce radiation to a negligible quantity, thus permitting routine treatments to be given, even in unshielded rooms, without attendant radio interference.

STUDIES OF HEAT SENSATION

In connection with the treatment of the accessory sinuses, we have recorded the facial heat sensations of 150 consecutive unselected patients. We followed the procedure of Kowarschik and Weisz (unpublished data cited by Mittelmann, 1938) in dividing the range of heat sensation into

four types: (1) threshold of warmth or barely perceptible heat, (2) feeling of definite warmth, (3) intensive warmth, (4) intolerable heat.

The data were obtained after completing the calibration of the dosimeter. A frequency of 42 megacycles was used. Electrodes with a diameter of 9 cm. were placed laterally against the face. The power input to the patient was reduced to zero and then gradually increased from the threshold until the higher heat sensations were observed. The results as given in the table agree fairly well with those published by Mittelmann (1938).

The table reveals wide variations in heat sensation and shows in a striking manner the unreliability of the empiric method of determining dosage. For different patients the threshold, for example, varies from 5 to 18 watts, a range of 350 per cent. The range producing the second degree of facial heat is similar. Thus, the use of heat sensation is an inexact measure of power input to the patient.

Facial Heat Sensations of One Hundred and Fifty Patients Treated with Ultrahigh Frequency Diathermy

Degree of Heat Sensation	Range in Watts	Average Watts	Mittelmann's Results in Watts
1.....	5 to 18	9	12
2.....	10 to 36	18	20
3.....	19 to 48	30	22
4	28 to 50	41	29

Our experience has indicated that a dose rate of 15 to 20 watts for a period of fifteen to twenty minutes is a safe and effective dose for the majority of patients. We believe that for the head 25 or more watts for this length of time is excessive. It is only occasionally that the patient's heat sensation builds up enough during treatment to warrant reduction of the input. When the temperature of the room rises above 74 F. or the temperature of the patient is over 100 F., sweating is apt to occur and should be dried frequently, as the drops of moisture tend to concentrate heating on small areas.

CONCLUSIONS

A complete description of treatment conditions requires that the dose rate, length of treatment, frequency or wavelength and applicator size and position be specified. A dosimeter such as the one described provides a simple accurate means of determining the dose rate and dosage of ultrahigh frequency diathermy. In the interests of safety and sound practice, all therapeutic machines should be equipped with some such means for measuring the power absorbed by the patient.

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LABORATORY ANALYSIS OF THE CONTENT OF TONSIL CRYPTS AS OBTAINED BY THE WET SUCTION TECHNIC

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There are numerous and divergent opinions concerning infection of the tonsil, but the majority seem to agree on the presence of cryptal plugs as a factor. Cryptal plugs are conducive to tonsillar infection because they block the passage through which the tonsil normally rids itself of cells and bacteria. They provide an environment of warmth, moisture, nutriment and varying degrees of oxygen tension, which is ideal for the growth, propagation and colonization of both aerobic and anaerobic organisms, and favor the retention and absorption of toxic metabolic products. Many of these, notably members of the Fusiformis group, are capable of causing necrosis in the deep epithelial lining of the crypt, with ensuing tonsillar infection. Aeration and circulation are interfered with, causing pressure on contiguous tissues and capillaries, a condition which may in turn produce congestion, inflammation and thrombosis.

In 1933, Dr. Otto Kahler [1] published an extensive review of the tonsil problem. Concerning cryptal plugs, he stated that: "Microscopic examination of the plugs, of which there are several kinds, is an important means of deciding the question whether the tonsils are diseased and may have caused general illness." In a large number of cases he examined the contents of crypts and found that the plugs from persons suffering with diseases presumably caused by the tonsils, such as articular rheumatism, endocarditis and nephritis, as well as those from patients suffering with relapsing angina, were, whether fluid or solid, extremely rich in leukocytes. This finding was present in 80 per cent of the cases, whereas in 20 per cent only a few leukocytes were found. In people who did not have infections of the tonsils plugs were by no means less

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1. Kahler, O.: The Tonsil Problem, J. Laryng. & Otol. **48**:2 (Jan.) 1933.

frequent, but the percentage proportions with respect to the presence of leukocytes were reversed. In 80 per cent the plugs were free from leukocytes, and in 20 per cent they were rich in leukocytes.

The unsatisfactory feature of most examinations is that they are made by the pathologist after the tonsil has been excised. If examination is carried out before the excision, it is usually by the exertion of light pressure, which fails to reveal conditions in the more important depths of the crypt. The information thus obtained either comes too late for the institution of preventive treatment or leads to an incorrect diagnosis.

Of the various methods used for ridding the tonsil of deleterious accretions, that of wet suction irrigation has proved the most efficient. It provides excellent opportunity for microscopic examinations of cryptal plugs during the process of treatment and obviates the necessity of excising the tonsil to ascertain the degree of infection.

This thesis is an attempt to demonstrate: (1) the therapeutic value of wet suction irrigation in the treatment of infected tonsils, (2) the mechanical superiority of the wet suction method over the original dry suction in evacuating the tonsillar crypts and (3) the value of cultural and microscopic examination of the centrifuged sediments of the return irrigation fluids as an adjunct to clinical diagnosis.

During the past eight years, we have used wet suction irrigation with physiologic solution of sodium chloride continuously in the treatment of diseased tonsils with uniformly satisfactory results. We have never used antiseptics; for we believe the basic principle underlying treatment of tonsillar infections is essentially that of eliminating accumulated accretions by mechanical cleansing. If this can be accomplished without mechanical trauma, the use of antiseptics is superfluous. We have seen patient after patient with acute follicular tonsillitis cured by two or three wet suction irrigations with physiologic solution of sodium chloride. In no instance have we observed spread of the infection or accentuation of clinical signs or symptoms following its use. We have merely tried to maintain the normal function of the tonsillar crypts by keeping them open and unobstructed. Once normal function is established, it is amazing how well the tonsil can deal with the most severe infections.

One of our cases was noteworthy. The patient was suffering from a large peritonsillar abscess, which cleared up completely within two days after two treatments by wet suction irrigation. No complications occurred, and there was no recurrence.

Both acute and chronic infections have been treated by this method, and it seems highly probable that acute attacks have sometimes been averted by resorting in time to the rational procedure of mechanical

cleansing. It is also quite evident that by this treatment surgical intervention has been rendered unnecessary in many cases in which ordinarily operation would have been indicated.

DESCRIPTION OF APPARATUS

Dry suction has long been a familiar form of therapy; one of the earliest and most widely known forms was used in the treatment of pulmonary congestion. An ordinary glass tumbler was swabbed on the inner surface with alcohol and ignited. This flaming cup was then placed over the back of the patient. The combustion of the alcohol exhausted the air content and created a vacuum. Suction thus induced caused local hyperemia of the underlying skin and, more often than not, ecchymoses or petechial hemorrhages.

After the development of the air pump, with its air and suction gage, refinements in the application and use of dry suction appeared. Glass tubes were developed to cover the buccal surface of the tonsil, and calibrated suction was applied as a means of combating tonsillar infection.

Experimental work [2] proved that dry suction applied in this manner withdrew the surface accumulation of the crypts if the suction was built up to 15 pounds (6.8 Kg.), but failed to empty the crypts. Suction under 15 pounds, on the other hand, failed to remove surface secretion. Considerable trauma both of the tonsil and of the adjacent structures was occasioned by using dry suction of 15 pounds. This was evidenced by increased congestion and spread of infection. Despite these drawbacks, the principle underlying the application of suction to the tonsil as a mode of therapy was sound.

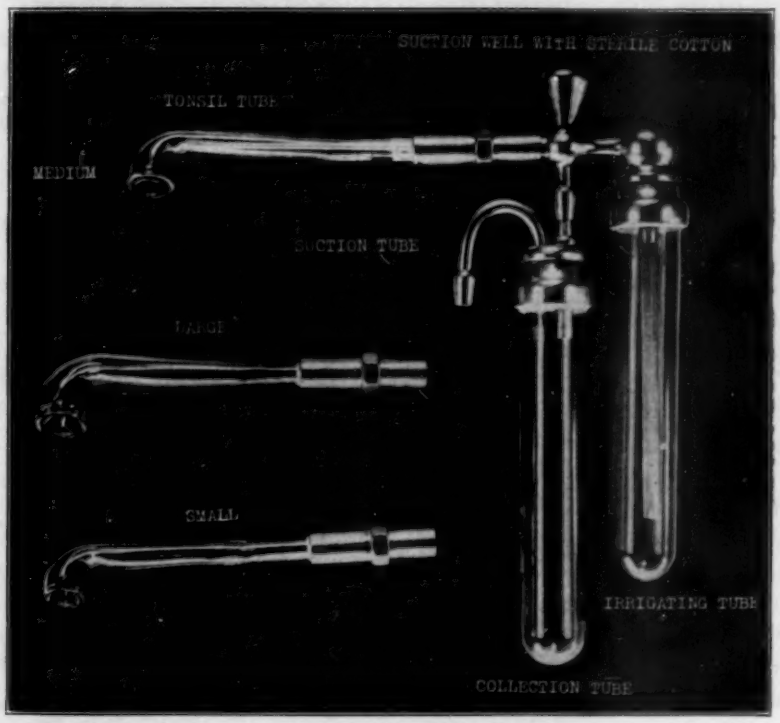
Further experiments carried out to discover some means to overcome these detrimental features led to the development of the wet suction tonsil instrument.[3] It was designed to apply direct suction to the tonsil as a whole and at the same time irrigate the tonsillar crypts.

It consists of a glass suction tube, of which there are three sizes to fit various tonsils, a finger control suction valve and a glass tube for the irrigating fluid; the parts are shown in the illustration. The tonsil tube is attached to the suction valve by means of a tight-fitting friction joint, permitting the tube to be rotated to engage either the right or the left tonsil. Running through the tonsil tube and suction valve is a small hollow metal tube, which conveys the irrigating fluid from the irrigating tube through the suction valve and tonsil tube over the tonsil and into the tonsillar crypts as soon as suction is applied. The application of

2. McAuliffe, G. W.: Wet Suction of the Tonsil, Eye, Ear, Nose & Throat Monthly **10**:325 (Sept.) 1931.

3. McAuliffe, G. W.: A Wet Suction Unit for Ear, Nose and Throat, Eye, Ear, Nose & Throat Monthly **11**:230 (July) 1932.

suction draws the tonsil outward into the tube, causes the crypts to open and allows the irrigating fluid to penetrate deeply into each crypt as the secretion is carried out. Suction opens the crypts and withdraws the plugs; irrigation softens and dissolves the harder particles and thoroughly cleanses the walls and fundus of each crypt. A twofold action thus takes place. Suction, the only positive force used, is counterbalanced by irrigation, which is directly proportionate to the suction used. A calibrated gage controls the required degree of suction before application, and placing the index finger over the top of the suction valve closes the suction gap after application.



Wet suction apparatus.

We proposed to arrive at a factual estimation of the efficacy of this method by comparing the bacteriologic and the cytologic observations of the specimen thus obtained with those of the excised tonsil. Would the bacterial flora of the crypts approximate that of the tonsil proper or merely that of the mouth? Would these accretions contain large numbers of leukocytes, as Kahler observed in infected tonsils? Finally, could any practical information be derived from the examination of these plugs? In an endeavor to answer these questions, we drew up the scheme of laboratory methods now to be described.

TECHNIC

Five specimens were taken from each patient as follows: (1) from the nasopharynx by means of a West tube swab, (2) from the tonsil surface by means of a swab, (3) from the sublingual saliva by means of a swab, (4) from the tonsil by irrigation with a wet suction tube in physiologic solution of sodium chloride and (5) from the excised tonsil.

This was done in order to differentiate as far as possible between the usually saprophytic so-called mouth organisms and colonized organisms thought to be causing infection, and further, to ascertain whether the flora recovered from irrigation sediments approximated that found in the oral cavity.

All specimens were taken while the patient was in the operating room immediately preceding tonsillectomy and sent directly to the laboratory, where cultures were made on their arrival. This routine was followed because we recognized not only the seasonal and environmental changes in bacterial flora but also the almost constant changes that occur to modify the presence of transient bacteria in the oral cavity,[4] and it is essential in comparative study that conditions be as equal and constant as possible.

Cultures were made of each specimen on two successive horse blood–agar streak plates and in two dilution plates. These were incubated at 37 C. (99 F.) for twenty-four hours and a preliminary reading made. They were returned to the incubator and examined after forty-eight hours.

Wet suction irrigation fluid collected in a sterile 50 cc. centrifuge tube interposed in the suction gap was immediately centrifuged; the supernatant fluid was discarded and the sediment cultured as described. Cultures were also made of the exudate from the excised tonsil. The same types of organisms were recovered from both specimens.

Cultures taken from the nasopharynx were usually similar, though the growth was often sparse, and Fusiformis organisms were absent. Cultures taken from the tonsil surface with a swab were unreliable; Streptococcus haemolyticus was not always recovered, and Fusiformis organisms were absent. Culture growths from the sublingual saliva were almost invariably Streptococcus salivarius and Staphylococcus albus. Fusiformis organisms were nearly always present in the irrigation sediment.

The results are shown in table 1.

In addition to the cultures, stained films were made from the irrigation fluid sediment and from the excised tonsil. Material expressed from the latter was rubbed up in a drop of physiologic solution of sodium chloride on new and freshly

4. Wheeler, G. W.; Wilson, M. G., and Leask, M. M.: The Relation of Upper Respiratory Infections to Rheumatic Fever in Children: III. The Seasonal Bacterial Flora of the Throat in Rheumatic and Non-Rheumatic Children, J. Clin. Investigation **14**:345 (May) 1935.

polished slides. Spreads were drawn out as in hematologic procedures. One of the slides was stained with the Hastings blood stain; the other, by the Gram method.

In making cell counts, the whole slide was first examined, and the general characteristics were noted. Then eight fields were selected, and the counts were recorded. Because we were primarily interested in the presence of polymorphonuclear leukocytes, the fields showing these cells were selected as far as possible for the counts. These counts are to be considered on a purely relative basis, their purpose being to show the preponderance of one type of cell over another in material obtained by two different methods.

It should also be clearly understood that we are not concerned in this paper with the histopathologic condition of the tonsil. Such studies are obviously made only by means of the pathologic tissue section, wherein the cytologic differentiation, topography and balance are revealed intact. We present an approximate cell count of exudate expressed by manipulation solely to afford visual evidence of the destruction resulting from pressure exceeding that of the 15 pound wet suction.

COMMENT

Bacteriologic Observations.—The organisms encountered in the irrigation sediments from 15 patients were as follows: Staphylococcus aureus, Streptococcus haemolyticus beta, Pneumococcus, Haemophilus influenzae, Streptococcus alpha and alpha prime, Streptococcus gamma, Fusiformis (various), diphtheroid bacilli, Micrococcus catarrhalis and Micrococcus pharyngis siccus.

No one organism can be considered a specific causative agent in all cases of tonsillitis. Individual susceptibility, seasonal prevalence, colonization within the crypts and many other conditions modify the bacterial flora from time to time. Nearly all the organisms enumerated are potential pathogens; yet they can sometimes lead a purely saprophytic existence within the crypt.

Although we were able to study only 15 cases in this series, we found Streptococcus haemolyticus beta in 93 per cent of these. Because of the pathogenicity and toxicity of this organism, we regard it with suspicion and feel that its presence in the clinically diseased tonsil is indicative of infection, especially when we note the frequency with which it is associated with members of the Fusiformis group. The result of symbiosis of these two types of organisms has been noted by many bacteriologists, and special reference is made to it by Smith in his work on oral spirochetes.[5]

Incidentally, it may be of interest to note that during the year 1939-1940, we made cultures from 93 tonsils from patients on the private side of the hospital. Of these 93 tonsils, 76 showed Streptococcus haemolyticus beta usually predominating, making a total of 81.8 per cent.

5. Smith, D. T.: Oral Spirochetes and Related Organisms in Fuso-Spirochetal Disease, Baltimore, Williams & Wilkins Company, 1932.

TABLE 1.—*Comparative Bacteriologic Study of Cultures to Show Close Similarity Between Bacterial Flora of Irrigation Fluids and That of Excised Tonsils*

Patient	Specimen*	Staphylococcus Aureus	Staphylococcus Albus	Streptococcus Haemolyticus Beta	Streptococcus Microaerophilus	Streptococcus Alpha	Streptococcus Alpha Prime	Streptococcus Gamma	Pneumococcus (Type)	Haemophilus Influenzae	Micrococcus Catarrhalis	Streptococcus Salivarius	Micrococcus Tetragenus	Micrococcus Pharyngis Siccus	Diphtheroid Bacilli	Fusiform Organisms
1	N.	+†	0	++	0	+	+	0	0	0	0	0	0	+	0	0
	T. S.	+	0	±	0	+	+	0	0	0	0	+	0	0	0	0
	S.	0	+	0	0	0	0	0	0	0	0	+	0	0	0	0
	I.‡	+	0	++	0	+	+	0	0	0	0	+	0	0	0	0
	E. T.	+	0	++	0	+	+	0	0	0	0	0	0	0	0	+
2	N.	+	0	0	0	+	+	0	0	0	0	+	0	0	0	0
	T. S.	+	0	0	0	+	0	0	0	+	0	+	0	0	0	0
	S.	+	0	0	0	0	0	0	0	0	0	+	0	0	0	0
	I.	+	0	+	0	+	+	0	0	+	0	0	0	0	0	+
	E. T.	0	0	+	0	+	+	0	0	+	0	0	0	0	0	0
3	N.	+	0	0	0	+	+	0	0	0	0	0	0	0	0	0
	T. S.	+	0	0	0	+	+	0	0	+	0	+	0	0	0	0
	S.	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0
	I.	+	0	+	0	+	+	0	0	+	0	+	0	0	0	+
	E. T.	+	0	+	0	+	+	0	0	+	0	0	0	0	0	0
4	N.	+	+	0	0	+	+	0	0	0	0	0	0	0	0	0
	T. S.	+	0	0	0	0	0	0	0	+	0	+	0	++	0	0
	S.	0	0	0	0	0	0	0	0	0	0	+	0	+	0	0
	I.	++	+	0	0	+	+	0	0	+	0	0	0	0	0	+
	E. T.	++	+	0	0	+	0	0	0	+	0	0	0	0	0	0
5	N.	++	0	0	0	+	+	0	0	0	0	0	0	+	0	0
	T. S.	++	+	0	0	+	+	0	0	0	0	0	0	+	0	0
	S.	+	0	±	0	0	0	0	0	0	0	0	0	0	0	0
	I.	++	0	+	0	+	+	+	0	0	0	+	0	0	0	0
	E. T.	++	0	0	0	+	+	0	0	0	0	+	0	0	0	+
6	N.	+	0	++	0	0	0	0	0	0	0	+	0	0	0	0
	T. S.	+	0	+	0	+	0	0	0	+	0	+	+	+	0	0
	S.	0	0	0	0	0	0	0	0	0	0	+	+	+	0	0
	I.	+	0	++	0	+	0	0	0	+	0	+	0	+	0	0
	E. T.	+	0	+	0	+	0	0	0	+	0	+	+	0	0	+
7	N.	+	0	+	0	+	0	0	0	+	0	0	0	0	+	0
	T. S.	0	+	0	0	0	0	0	0	+	0	+	0	0	+	0
	S.	0	0	0	0	0	0	0	0	0	0	+	0	0	+	0
	I.	+	+	++	0	+	+	+	0	+	0	0	+	0	+	+
	E. T.	+	0	++	0	+	+	0	0	+	0	0	+	0	+	0

8	N.	+	0	±	0	+	0	0	0	0	0	0	+	+	0	0
	T. S.	+	0	±	0	+	0	0	0	0	0	+	0	+	0	0
	S.	+	0	0	0	0	0	0	0	0	0	+	0	+	0	0
	I.	+	0	++	0	+	+	0	XIX	0	0	0	0	+	+	+
	E. T.	+	0	0	0	+	+	0	XIX	0	0	0	0	0	+	0
9	N.	0	0	±	0	0	+	0	0	0	0	0	0	0	0	0
	T. S.	+	0	±	0	+	+	0	0	+	0	+	0	0	0	0
	S.	0	+	0	0	0	0	0	0	0	0	+	0	0	0	0
	I.	+	0	+	0	+	+	0	0	+	0	0	+	0	0	+
	E. T.	+	0	+	0	+	+	0	0	+	0	0	+	0	0	0
10	N.	0	+	±	0	0	+	0	0	0	0	0	0	+	0	0
	T. S.	+	0	+	0	0	+	0	0	0	0	0	0	+	0	0
	S.	+	0	0	0	0	+	0	0	0	0	0	0	+	0	0
	I.	+	0	++	+	0	+	0	0	0	0	0	0	0	0	+
	E. T.	+	0	++	0	0	+	0	0	0	0	0	0	0	0	0
11	N.	+	0	+	0	0	+	0	0	+	0	0	0	0	0	0
	T. S.	+	0	±	0	+	+	0	0	+	0	0	0	+	0	0
	S.	±	0	0	0	0	0	0	0	0	0	+	0	+	0	0
	I.	+	0	+	0	+	+	0	0	+	0	0	+	+	0	+
	E. T.	+	0	+	0	+	+	0	0	+	0	+	+	+	0	0
12	N.	0	0	±	0	0	0	0	XVIII	±	0	0	0	0	0	0
	T. S.	+	0	0	0	0	+	0	0	+	0	+	0	0	0	0
	S.	0	+	0	0	0	0	0	0	0	0	+	0	0	0	0
	I.	+	0	+	0	+	0	+	XVIII	+	0	0	0	0	0	+
	E. T.	+	0	+	0	+	0	0	XVIII	+	0	+	0	0	0	0
13	N.	0	+	+	0	+	+	0	0	0	0	0	0	+	0	0
	T. S.	+	0	+	0	+	+	0	0	0	0	0	0	+	0	0
	S.	0	+	0	0	0	0	0	0	0	0	+	0	+	0	0
	I.	+	0	++	0	+	+	0	0	0	0	0	0	+	0	+
	E. T.	+	0	++	0	0	0	0	0	0	0	0	0	+	0	0
14	N.	+	0	+	0	0	0	0	II	0	+	0	0	0	0	0
	T. S.	+	0	0	0	0	0	0	II	0	0	+	0	++	0	0
	S.	+	0	0	0	0	0	0	0	0	0	++	0	+	0	0
	I.	+	0	+	0	+	+	+	II	0	+	0	0	0	0	+
	E. T.	+	0	+	0	+	+	+	II	0	+	0	0	0	0	0
15	N.	0	+	+	0	+	+	0	III	0	0	0	0	+	0	0
	T. S.	0	+	±	0	+	+	0	III	0	0	0	0	+	0	0
	S.	0	0	0	0	0	0	0	0	0	0	+	0	+	0	0
	I. Omitted															
	E. T.	+	0	+	0	+	+	+	III	0	0	0	0	+	0	0
Percentage of the 15 cases showing given organism		100	0	93	0	0	0	0	27	53.3						

* N., specimen from nasopharynx; T.S., specimen from tonsil surface; S., specimen from sublingual saliva; I., specimen from tonsil obtained by irrigation with wet suction tube; E. T., specimen from the excised tonsil.

† ++, heavy growth; +, moderate growth; ±, an occasional colony.

‡ Note similarity of I. and E. T.

It is our opinion that the best way to determine the bacterial flora of the tonsil before tonsillectomy is by cultures of wet suction irrigation sediments. It has been demonstrated (table 1) that the bacterial flora of these sediments is practically identical with the flora of the excised tonsil, whether this is compressed or ground up in a mortar. Therefore, the bacterial flora of the tonsil is the bacterial flora of the crypt.

This information cannot be derived with complete accuracy if the specimens are taken by means of a tonsil swab, because the depth of the crypt is not reached by a surface swab. Organisms such as M. pharyngis siccus and Str. salivarius are apt to predominate in the cultures; Streptococcus haemolyticus is sometimes, and Fusiformis nearly always, missed.

The work of Bloomfield [6] concerning the bacterial flora of the mouth and the fate of the organisms unable to colonize on the tonsil surface is of particular interest in this respect.

Cytologic Observations.—Together with cholesterol crystals, mucus, and detritus composed of dead and disintegrated matter, the cells encountered in these sediments (table 2) are, in their order of predominance, as follows: polymorphonuclear leukocytes, erythrocytes, epithelial cells, eosinophils and plasma cells. There were also a few lymphocytes, the latter resulting from lymphoid infiltration of the epithelium and derived apparently also from the blood, as they appeared in far greater numbers in irrigations in which slight hemorrhage accidentally took place before irrigation.

Table 2 shows a high percentage of polymorphonuclear leukocytes in nearly all cases examined. Pathologic sections of infected tonsils show these cells to be largely situated about the epithelium lining the crypts. The cryptal content is washed out by means of wet suction irrigation. The presence of polymorphonuclear leukocytes is indicative of infection. We refer, of course, to mature, actively phagocytic cells, not to be confused with disintegrated epithelial cells and leukocytes, which the tonsil discharges under normal functional conditions, dependent on lymphoid infiltration of the epithelium. We have found, as Kahler did, by means of microscopic examination, that the whitish secretion which can often be expressed from the tonsil crypt is not of necessity leukocytic pus. In some tonsils, e. g., the hypertrophic tonsils of young children during periods of endocrine activity, the exudate is mostly tissue lymph. We have examined thick creamy material

6. Bloomfield A. L.: The Fate of Bacteria Introduced into the Upper Air Passages, Am. Rev. Tuberc. **3**:553 (Nov.) 1919; Adaptation of Bacteria to Growth on Human Mucous Membranes, Bull. Johns Hopkins Hosp. **33**:61 (Feb.) 1922; Dissemination of Bacteria in Upper Air Passages, ibid. **33**:145 (April); 252 (July) 1922.

TABLE 2.—*Cells in Irrigation Fluid and Excised Tonsil Exudates*

Patient	Specimen	Number of Cells in 8 Fields	Fusiform Organisms	Spirochetes	Polymorphonuclear Leukocytes	Small Lymphocytes	Large Lymphocytes	Lymphoblasts	Plasma Cells	Eosinophils	Mast Cells	Erythrocytes	Endothelial Leukocytes	Epithelial Cells	Miscellaneous Large Monocytes	Cholesterol Crystals	Percentage of Polymorphonuclear Leukocytes	Comment
1	Irrigation fluid	20	++	+	10	0	0	0	0	0	0	0	0	7	0	0	50	Recurrent sore throat; streptococcic injection twice; chronic tonsillitis
	Tonsil exudate	609	±	0	3	510	22	3	14	0	1	46	1	6	3	0	0.5	
2	Irrigation fluid	507	++	++	166	11	0	0	2	3	0	301	0	20	4	0	32.7	Recurrent sore throat and palpable glands; low grade fever; chronic perifollicular tonsillitis
	Tonsil exudate	182	0	0	3	144	6	0	5	0	1	9	2	10	2	0	1.7	
3	Irrigation fluid	43	++	++	15	1	0	0	0	0	0	6	0	21	0	+	35	Recurrent sore throat; occasional otalgia; chronic tonsillitis
	Tonsil exudate	181	±	0	2	123	11	2	6	0	0	26	4	6	1	0	1.1	
4	Irrigation fluid	32	+++*	+++	11	0	0	0	0	0	0	0	0	21	0	+	34	Chronic cough; loss of weight and glandular enlargement; occasional sore throat; chronic tonsillitis (cause of cough?)
	Tonsil exudate	693	±	0	5	472	31	13	14	3	2	144	0	9	0	0	0.72	
5	Irrigation fluid	110	+++	+++	64	0	0	0	0	0	0	0	0	46	0	+	59	Chronic catarrhal otitis media; no sore throat or enlargement of glands; normal temperature; subacute tonsillitis
	Tonsil exudate	301	0	0	8	85	3	5	7	6	0	154	2	30	1	0	2.7	
6	Irrigation fluid	47	++	+	23	1	0	0	0	0	0	3	0	20	0	0	49	Recurrent sore throat and palpable glands; no ear difficulty; impaired nasal breathing; chronic tonsillitis
	Tonsil exudate	414	+	0	11	244	21	8	3	2	1	107	0	12	5	0	2.6	
7	Irrigation fluid	83	+++	+++	28	0	0	0	0	0	0	30	0	23	1	0	34	Recurrent sore throat; otalgia; frequent colds with glandular involvement; chronic tonsillitis
	Tonsil exudate	756	0	0	5	635	14	3	23	0	1	53	0	4	18	0	0.65	
8	Irrigation fluid	32	++	++	15	0	0	0	0	0	0	5	0	12	0	0	47	Recurrent sore throat; 1 attack of quinsy; chronic perifollicular tonsillitis
	Tonsil exudate	528	±	0	11	381	26	2	20	2	2	75	0	9	0	0	2.2	
9	Irrigation fluid	...	.....	.....	...	...	Irrigation omitted				..	...	..	..	..	..	....	Slight elevation of temperature and loss of weight; leukocytosis; chest normal; cheesy material from tonsils on traction; no fibrous tissue; chronic tonsillitis
	Tonsil exudate	527	±	0	10	373	29	10	12	2	1	78	6	5	1	+	1.9	
10	Irrigation fluid	53	+	+	22	0	0	0	0	0	0	19	0	12	0	+	41.5	Recurrent sore throat; slight elevation of temperature; beginning arthritis; chronic tonsillitis
	Tonsil exudate	478	0	0	13	400	26	2	22	1	1	8	0	5	0	+	2.7	
11	Irrigation fluid	254	+++	+++	26	1	0	0	0	0	0	208	0	19	0	+	10.23	Cervical adenitis; recurrent sore throat; occasional otalgia; chronic tonsillitis
	Tonsil exudate	520	0	0	10	401	22	19	15	0	1	29	0	23	0	0	1.9	
12	Irrigation fluid	101	+++	+++	43	0	0	0	0	0	1	40	0	17	0	0	42.57	Recurrent sore throat; beginning arthritis; chronic tonsillitis
	Tonsil exudate	627	0	0	6	443	8	25	9	0	1	102	2	31	0	0	0.95	
13	Irrigation fluid	78	++++	++++	49	0	0	0	0	0	0	13	0	16	0	0	62.8	Recurrent sore throat beginning polyarthritis; infected tonsils
	Tonsil exudate	389	±	0	7	282	13	3	12	0	0	45	0	27	0	0	1.8	
14	Irrigation fluid	104	++++	++++	51	0	0	0	0	2	0	10	0	41	0	0	49	Chronic tonsillitis with specific focus
	Tonsil exudate	411	0	0	3	289	12	3	12	0	0	69	1	22	0	0	0.73	
15	Irrigation fluid	...	.....	.....	...	...	Irrigation omitted				..	...	..	..	..	..	....	Recurrent sore throat; chronic discharge from ear; simple mastoidectomy year previous; cervical adenitis; no history of sinusitis; chronic tonsillitis
	Tonsil exudate	287	+	0	1	259	16	3	4	0	0	0	0	3	1	0	0.35	

* +++, numerous; ++++, very numerous.

expressed from so-called pus pockets, situated about the pharyngopalatine arch, suggesting peritonsillar abscesses, which on microscopic examination proved to be but a solid mass of epithelial cells.

Numerous erythrocytes were found in some of these films. Their presence can be accounted for, however, by the fact that when a general anesthetic is given preparatory to tonsillectomy, the pharynx, nasopharynx and hypopharynx must be cleared of secretion by means of dry suction. Dry suction, as we have seen, invariably causes some bleeding, particularly in the pharynx and over the posterior pillars of the tonsil. In cases in which dry suction was not permitted, only a few erythrocytes appeared.

The desquamated epithelial cells in these preparations are of no particular interest. They are found in the crypts as on the surface of any inflamed mucous membrane.

Eosinophils were found only occasionally. One case was an interesting exception, with a percentage of 13.8. Unfortunately, we are unable to offer any explanation for the presence of these cells in the irrigation fluid, owing to the extremely limited history provided by the patient, who was referred to us by a physician from the South.

Plasma cells, although numerous in chronically inflamed lymphoid tissues, are apparently not present to any great extent within the tonsillar crypts.

Trauma.—We were naturally concerned as to whether or not the use of wet suction caused trauma of the tonsil. The procedure is practically painless, and local anesthesia has never been required. The fact that we have dealt with acutely inflamed tissue without undue discomfort to the patient by this method is supportive evidence that pain is not a factor. There is unfortunately no laboratory test available to prove or disprove the contention as to trauma. Microscopic examination of irrigation sediments shows no connective tissue or reticular cells. It does show a marked absence of lymphocytes as opposed to the large numbers seen in exudate forced up through the crypts by manipulating the tonsil. The parenchyma of the tonsil consists essentially of lymphoid cells. Practically no lymphocytes are found in these preparations that could not be attributed to the results of lymphoid infiltration of the epithelium. It seems reasonable, therefore, to assume that wet suction at 15 pounds has no destructive effect on the lymphoid structure of the tonsil and that its action is restricted to the crypt. Under higher pressures the epithelium is ruptured, resulting in damage to the whole structure and spread of infection.

The count of epithelial cells is relatively high. This cannot, however, be attributed to mechanical injury to the squamous epithelium lining

TABLE 3.—*Cell Counts on a Series of 37 Irrigation Fluids*

TABLE 3.—*Cell Counts on a Series of 37 Irrigation Fluids*

Patient	Tonsil	Number of Cells in 8 Fields	Fusiform Organisms	Spirochetes	Polymorphonuclear Leukocytes	Small Lymphocytes	Large Lymphocytes	Lymphoblasts	Plasma Cells	Eosinophils	Mast Cells	Erythrocytes	Endothelial Leukocytes	Epithelial Cells	Miscellaneous Large Monocytes	Cholesterol Crystals	Percentage of Polymorphonuclear Leukocytes	Comment
1	Right	420	++	+	337	0	0	0	1	6	0	28	0	48	0	0	77	
	Left	240	++	+	174	0	0	0	0	3	0	28	0	35	0	0	72	
2	Right	117	+++	+++	66	0	0	0	0	0	0	26	0	25	0	0	56	
	Left	57	+++	+++	41	0	0	0	0	0	0	2	0	14	0	0	72	
3	Right	117	+++	+++	93	0	0	0	0	0	0	2	0	22	0	0	80	
	Left	84	+++	+++	66	0	0	0	0	0	0	5	0	13	0	0	78	
4	Right	181	++	++	33	4	3	0	0	0	0	126	0	15	0	0	18.2	
	Left	90	++	++	26	4	0	0	0	0	0	30	2	28	0	0	29	
5	.....	287	+++	+++	48	20	6	1	0	0	0	200	0	10	0	0	16.7	Bleeding from West tube swab
6	Right	98	+++	+++	71	0	0	0	0	0	0	8	0	19	0	0	72	
	Left	118	+++	+++	75	0	0	0	0	3	0	15	0	25	0	0	63.5	
7	First	86	+++	+++	61	1	1	0	1	5	0	5	0	12	0	0	71	
	Second	35	+++	+++	25	0	0	0	0	0	0	2	0	8	0	0	71.5	
8	First	81	+++	+++	61	4	3	0	2	0	0	11	0	0	0	0	75.3	
	Second	..	++++	++++	0	Almost complete absence of cells—masses of fusiform organisms and cocci												
9	Right	80	+++	+++	49	0	0	0	0	0	0	10	0	21	0	0	61	
10	.....	20	++	+	10	0	0	0	0	0	0	0	0	10	0	0	50	
11	.....	507	++	++	166	1	0	0	2	3	0	301	0	30	4	0	32.7	
12	.....	43	++	++	15	1	0	0	0	0	0	6	0	21	0	+	51	
13	.....	32	+++	+++	11	0	0	0	0	0	0	0	0	21	0	+	34	
14	.....	110	+++	+++	64	0	0	0	0	0	0	0	0	46	0	+	59	
15	.....	47	++	+	23	1	0	0	0	0	0	3	0	20	0	0	49	
16	.....	83	+++	+++	28	0	0	0	1	0	0	30	0	23	1	0	34	
17	.....	53	+	0	22	0	0	0	0	0	0	19	0	12	0	+	41.5	
18	.....	254	+++	+++	26	1	0	0	0	0	0	208	0	19	0	0	10.23	
19	.....	101	+++	+++	43	0	0	0	0	0	1	40	0	17	0	0	42.57	
20	.....	78	++++	++++	49	0	0	0	0	0	0	13	0	16	0	+	62.8	
21	.....	104	++++	++++	51	0	0	0	0	2	0	10	0	41	0	0	49	
22	Right	126	++	++	81	0	0	0	0	16	0	25	0	4	0	0	64.3	
	Left	7	++++	++++	5	0	0	0	0	0	0	1	0	1	0	0	71.4	
23	Right	33	+	0	8	0	0	0	0	0	0	3	0	22	0	0	24	
	Left	105	+	0	54	0	0	0	0	1	0	27	0	23	0	0	51.5	
24	.....	101	+++	+++	43	0	0	0	0	0	1	40	0	17	0	0	42.57	
25	.....	139	++	+	67	0	0	0	1	0	0	51	0	20	0	0	48.2	
26	.....	115	+++	+++	91	0	0	0	0	1	0	3	0	20	0	0	79.1	
27	.....	152	++	+	86	3	0	0	0	21	0	37	0	5	0	0	56.6	Eosinophils, 13.8%
28	.....	387	++	0	378	0	0	0	0	4	0	2	0	3	0	0	97.7	

the crypt, since there occurs a natural desquamation of epithelial cells in any inflamed mucous membrane. The presence or absence of these cells in the irrigation fluid is therefore of little significance.

SUMMARY

In presenting this brief investigation of cryptal contents, we wish to stress the fact that we do not presume to offer an infallible remedy for the relief of the victim of tonsillitis or a specific laboratory procedure for the positive diagnosis of focal infection. The tonsil problem has always been and still is bewildering. The clinical point of attack is uncertain because the mechanism and function of the tonsil itself are obscure and because the infective agent and the route by which it assails the tonsil are problematic.

Wet suction irrigation has proved a beneficial mode of therapy. The microscopic examination of these fluids is a step in the right direction for observing the trend of infection in the crypts, but the final diagnosis still rests on a variety of factors. The most important of these is the condition of the nasal sinuses [7]; also significant are the clinical appearance, individual anatomy, history, age, status of endocrine function and general condition of the patient.

We wish to see the tonsil regarded not as an independent and isolated organ but as a small, though highly important part of the whole lymphatic system, to be treated squarely and broadmindedly in accordance with this concept.

CONCLUSIONS

1. Wet suction at 15 pounds is an effective means of ridding the tonsillar crypts of an accumulation of organisms, cells and debris, thereby relieving congestion and inflammation

2. Wet suction at 15 pounds exerts no action beyond the epithelial lining of the tonsillar crypts.

3. The bacterial flora of an infected tonsil can be ascertained accurately before tonsillectomy by making cultures of the sediment of centrifuged irrigation fluids.

4. Cytologic examination of these sediments is helpful in differentiating between hypertrophy of the tonsil due to infection and that due to normal endocrine activity.

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7. Griffiths, I.: The Function of the Tonsils and Their Relation to the Aetiology and Treatment of Nasal Catarrh, Lancet **2**:723 (Sept. 25) 1937.

POSITION MALIGNANCY

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Some tumors, benign as to cell type, might be classified as malignant if by their position or location they constitute a threat to life or to vital anatomic structures. The heterogeneous, rather benign tumors to be described here deserve to be grouped together only because of this so-designated position malignancy and because of their common benign cellular structure. Most of them are rather rare or at least unusual.

REPORT OF CASES

CASE 1.—Mr. B. B., 23 years old, was first examined in 1925, because of a discharge from the right ear, which had been present since childhood. The personal history and the marital history were noncontributory. Two small perforations were seen in the drum membrane—one anterior and low, near the eustachian tube; the other high, in Shrapnell's membrane. There was a scanty purulent discharge, and a small granulation protruded through the upper perforation. There was considerable middle ear deafness, and roentgenograms showed sclerosis throughout the right mastoid process. The left ear, the nose and sinuses and the throat were essentially normal. The patient was given conservative treatment and was kept under observation. In 1926 and again in 1927 aural polyps were removed.

In August 1931 this patient came to me complaining of severe headache and requested examination of his ear, since he had been warned to have his ear examined if there were any unusual symptoms. He had had some nausea and vomiting for twenty-four hours. The ear was examined, with roentgen study, and was found as before. The eyes were examined by an oculist, who found a high error of refraction. New glasses were prescribed, and the fundus was reported to be normal. A neurologic examination gave negative results. In a few days the headache and vomiting ceased, and the patient lost no time from his work.

On September 20 he again reported for examination, with an attack of headache, nausea and vomiting. The conditions observed on aural examination were the same as those observed six weeks before. The attack lasted two days, and again he returned to work feeling well.

In October he again returned for examination because of headaches. During the latter part of October he lost a few days from work because of a severe headache which came on after an automobile ride.

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On November 5 he was admitted to the hospital, suffering with excruciating headache. The conditions observed on examination of the ear were as before—two small perforations in the right ear drum, with a scanty foul discharge. The hearing tests again showed a middle ear type of deafness with some useful cochlear function. Labyrinthine tests were done, and all responses were present, although somewhat delayed. Examination of the eyes at this time showed slight choking of both disks and small hemorrhagic streaks. Neurologic and medical examinations showed essentially normal conditions. Lumbar puncture was done. The spinal fluid revealed two cells, no increase in albumin or globulin and a normal sugar content. The Wassermann reaction of the spinal fluid was negative.

On November 6 a radical mastoidectomy was done. A wide surgical exposure of the lateral sinus and the dura was made over both the posterior and the middle fossa. No evidence was found which would lead one to suspect the presence of brain abscess or of meningitis from an aural infection. After the operation the pulse rate dropped at one time to 60 but quickly became normal and remained normal. A normal convalescence followed the operation. Repeated neurologic and medical examinations gave negative results. The patient went back to work two weeks after his operation, feeling well.

On December 7 he was readmitted to the hospital on account of severe headaches and remained for four days. Several times during this stay in the hospital the pulse rate was 60. The headache subsided, and the patient went back to work. Again medical and neurologic examinations gave no clue to the cause of the headache.

On December 24 the patient was readmitted to the hospital, with a pulse rate of 60, continuous vomiting and slight choking of the disks. The administration of morphine was necessary, to control the severe headache and restlessness. On December 25 he became unconscious. The white blood cells numbered 21,000. The spinal fluid showed 14 cells, with a slight increase of albumin and globulin and a normal sugar content. Respiration was very slow and irregular. The mastoid wound was revised, and a search was made in both the cerebellum and the temporosphenoidal lobe for a brain abscess, but no pathologic condition was found. At about 8 p. m., December 26, respiration ceased, but artificial respiration was carried on for almost three hours, the patient's heart continuing to beat for this time. Death occurred at about 11 p. m.

A variety of clinical diagnoses were considered: (1) brain abscess, (2) encephalitis and (3) brain tumor. It was thought that death was due to respiratory failure and increased intracranial pressure.

Pathologic Examination of the Brain.—"The specimen was received Dec. 28, 1931 (postmortem examination, Dec. 26, 1931).

"Gross Observations: There is a tumor mass of the choroid plexus the size of a small cherry (12 mm. in diameter), which is brownish purple, smooth and tense and shows several vascular attachments to the plexus. It lies in the third ventricle and is freely movable, changing its position readily, so that it can block either the foramen of Monro or the aqueduct of Sylvius. The third ventricle is much enlarged and there is an internal hydrocephalus—both lateral ventricles being enlarged and distended. Midsection shows the mass to be solid, with a fibrous tissue wall, and filled with a brownish colloid material, which shells out of the limiting wall.

"Microscopic Observations: There is a thick laminated wall of connective tissue which shows multiple attachments to the ependymal and vascular layers

of the choroid plexus. The major portion of the contents of the tumor appears as solid granular material, with an occasional small mononuclear cell and numerous open smooth fissures, appearing like cholesterol clefts. Nearer the limiting membrane these clefts are seen to be lined by cuboid or low pyramidal cells with large pyknotic nuclei and acidophilic cytoplasm. Between the clefts are numerous similar cells embedded in the solid granular interstitial substance. Psammoma bodies are abundant throughout the choroid plexus but not in the tumor mass or its envelopes.

"Diagnosis: Degenerated cholesteatomatous ependymal cyst of the third ventricle."

Comment.—This case furnishes a typical example of how a relatively simple and benign cellular tumor attains malignancy by position. Usually, ependymomas are found in children, and they may arise from the choroid plexus of any of the ventricles. They vary greatly in size. In 1936 Friedman and Solomon [1] listed 34 cases reported in the literature. Sixteen patients were over 15 years of age; the ages of 4 were unknown and 14 patients were under 15 years of age. These authors stated cyst formation may occur and may leave the true tumor a mere nodule in the wall of the cyst. The cysts vary greatly in size, and, as suggested by Davis and Cushing.[1a] secretory activity of the cells of the tumor causes the cyst formation.

They have been stated to occur most commonly in the fourth ventricle,[2] next in the lateral ventricles and least frequently in the third ventricle. The tumor tissue first assumes a papillary structure, the core of which may undergo myxomatous degeneration.

Jefferson and Jackson,[3] who reported 3 choroid cysts of the third ventricle, stated that ependymal rests long have been known to lie close beside the ventricles and were believed to be the possible starting point for cysts or solid tumors. The tumors produce no constant localizing signs; the effects are the result of obstruction of the cerebrospinal fluid alone. The symptoms are notoriously intermittent, and at times certain postures of the head can change or cause abatement of the headache, possibly by so changing the position of the tumor that the cerebrospinal fluid can again flow through the interventricular foramens. Both Jefferson and Jackson [3] and Davidoff and Dyke reported epithelial cysts of a dermoid type, with thick cheesy yellow contents, occurring in the ventricles, with origin in the choroid plexus.

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1. Friedman, J. C., and Solomon, C. I.: Tumors of the Choroid Plexus in Childhood, Am. J. Dis. Child. **52**:114 (July) 1936.

1a. Davis, L. E., and Cushing, H.: Papillomas of the Choroid Plexus with a Report of Six Cases, Arch. Neurol. & Psychiat. **13**:710 (June) 1925.

2. Weinstein, E. A.: Ependymomas Arising from the Ventricular Lining and the Choroid Plexus, J. Mt. Sinai Hosp. **5**:573 (Nov.-Dec.) 1938.

3. Jefferson, G., and Jackson, H.: Tumors of the Lateral and of the Third Ventricle, Proc. Roy. Soc. Med. **32**:1105 (July) 1939.

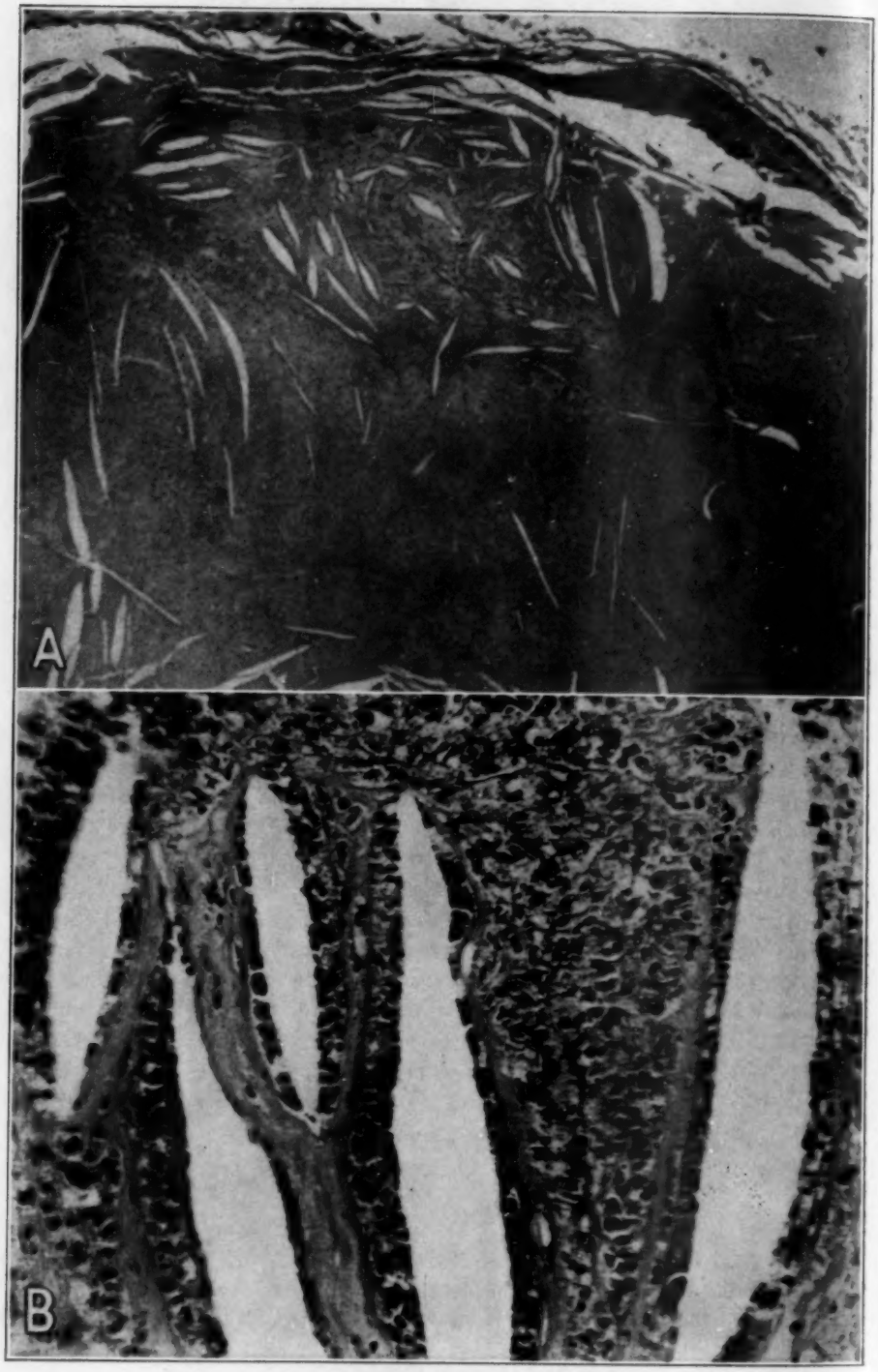

Figure 1

(See legend on opposite page)

CASE 2.—Mr. H. C., 21 years old, a textile worker, came for examination in March 1936, complaining of difficulty in swallowing and of a lump in his throat, which he realized had been present and growing for the last four years. Examination of the ears and nose gave entirely negative results. He had had a clean tonsillectomy. Involving the left lateral pharyngeal wall, extending from just behind the posterior pillar (at the lower pole of the left tonsil) to the piriform sinus, was a tumor the size of a hen's egg. It was firm and did not seem to be fixed, although there was difficulty in manipulating a tumor of this size in this area. There was no involvement of the cervical glands. The diagnoses considered preoperatively were: (1) Hodgkin's disease, (2) parotid tumor and (3) fibroma.

Operation.—On March 18, with the patient under anesthesia induced with ether, the tumor was removed through the mouth. By virtue of its size it extended deep into the neck, and although the removal was relatively easy and bloodless, because there was a well defined capsule, the carotid artery and the jugular vein were easily identified through the thin connective tissue of the capsule after the removal of the tumor.

Pathologic Examination of Tumor from the Throat.—"Gross Observations: The tumor weighs 30.8 Gm. and measures 6 by 4.5 by 2.5 cm. It is smooth and encapsulated, without rupture of the capsule. Midsection shows a solid soft edematous tissue.

"Microscopic Observations: All the areas examined show a solid fibrillar structure arranged in lamellae, with scanty fusiform nuclei and abundant fine fibrillae in wavy strands and irregular masses. Masson's trichrome and Foote's trichrome stains [3a] demonstrate a large neurogenic element, with the connective tissue confined largely to the dense capsule and invaginating septums along the abundant thin-walled vessels.

"Diagnosis: Benign neurofibroma."

Further Course.—The patient had an uneventful recovery. The rather large bed of the tumor filled in with clean granulation tissue and scarred over, with no resulting deformity. He was seen at intervals, and in September 1939 he had no sign of recurrence.

Comment.—I felt that the tumor was probably benign, and inasmuch as it was recovered entire without a rupture of the capsule, postoperative irradiation of the site was not advised. Although this operative approach to the tumor resulted in successful removal, on reconsideration I feel that

3a. Foot, N. C.: Peripheral Neurogenic Tumors, Am. J. Clin. Path. **6**:1 (Jan.) 1936; The Masson Trichrome Staining Methods in Routine Laboratory Use, Stain Technol. **8**:101, 1933. Masson, P.: Experimental and Spontaneous Schwannomas (Peripheral Gliomas), Am. J. Path. **8**:367 (July) 1932.

EXPLANATION OF FIGURE 1

Fig. 1 (case 1).—*A*, low power photomicrograph of a quadrant of the tumor, showing connective tissue limiting the capsular membrane which was attached to the choroid plexus of the roof of the third ventricle. Large numbers of "cholesterol clefts" are seen in the solid granular contents. *B*, high power photomicrograph of several clefts of the choroid cyst, lined by ependymal cells, which are seen in varying stages of degeneration in the solid substance between the clefts.

tumors such as this might be removed more safely by an external approach through the neck or at least after exposure of the great vessels of the neck, so that prompt ligation could be done in case uncontrollable hemorrhage was encountered. A tumor mass such as this, even though considered benign clinically, must be considered malignant by position if its close relation to the great vessels of the neck is weighed.

In the literature are descriptions of neurofibromas occurring in many different parts of the body. Penfield [4] defined a perineurial fibroblastoma as

> an encapsulated tumor which arises from the sheath of spinal nerve roots, cranial nerves, and peripheral nerves. It appears as a solitary tumor, much more often in a central location than in a peripheral one. Tumors of the acoustic and spinal nerve roots far outnumber all others. They do not metastasize, but may recur locally unless the segment of the attached nerve is also removed. The growth is usually slow. Sarcomatous degeneration has been reported, but infrequently.

In the literature at my disposal I have been able to find but a single somewhat similarly located tumor described. Malan [5] reported the removal of a neurofibroma the size of a walnut from under the right aryepiglottic fold. This tumor was removed by laryngofissure. Microscopic examination revealed a perineural fibroma.

Williams and Pastore [6] cited Desjardins as stating that neurofibromas are radioresistant.

CASE 3.—On March 6, 1931 C. Y., a girl 3 years old, was brought for examination. The mother said that since this little girl was 6 months old there had been some swelling above and in front of the right ear and that when she was a small child the orbital tissues were swollen. She had had four operations, which consisted, as nearly as I could determine, of curettage, incision and drainage. Back of the right ear, in the mastoid region, were two large draining fistulas, and in the swollen right temporal area were two deep scars. The posterior-superior wall of the right canal was sagging, and the ear drum could not be seen. The left drum was normal, and with the exception of enlargement and hypertrophy of the tonsils and adenoids the results of her examination were negative. Roentgenograms taken at this time showed "a destructive process throughout the entire right mastoid process, which obliterated practically all the mastoid structures." Because of my inability to obtain the cooperation of the terrified child, hearing tests and tests of the labyrinth were not done.

Operation.—On March 24, with the child under general anesthesia, a wide incision was made, exposing all of the postauricular area and the squamous

4. Penfield, W., cited by Stough, J. T.: Intra-Ocular Neurofibroma, Arch. Ophth. **18**:540 (Oct.) 1937.

5. Malan, A.: Neuronoma of the Larynx, Valsalva **13**:417 (Oct.) 1937; abstracted, Ann. Otol., Rhin. & Laryng. **47**:1135 (Dec.) 1938.

6. Williams, H. L., and Pastore, P. N.: Neurofibroma of the Facial Nerve: Destruction of Labyrinth and Mastoid Process, Arch. Otolaryng **29**:977 (June) 1939.

portion of the temporal bone under the temporal muscle. Thick gelatinous tissue, resembling granulation tissue, protruded through the bone. This mass of tissue was removed. A radical mastoidectomy was done, with removal of this granulation-like tissue as far as possible in every direction. The bony covering of the facial nerve had been destroyed, and the nerve was exposed at the distal end of the ridge. In both the horizontal and the superior canal there was a fistula. The destructive process had exposed the dura over the cerebellum and middle fossa, and the supposed granulation tissue was cleaned out also over a large area anteriorly located in the squamous portion of the temporal bone. Because of the unusual character of the tissue removed and because of the great amount

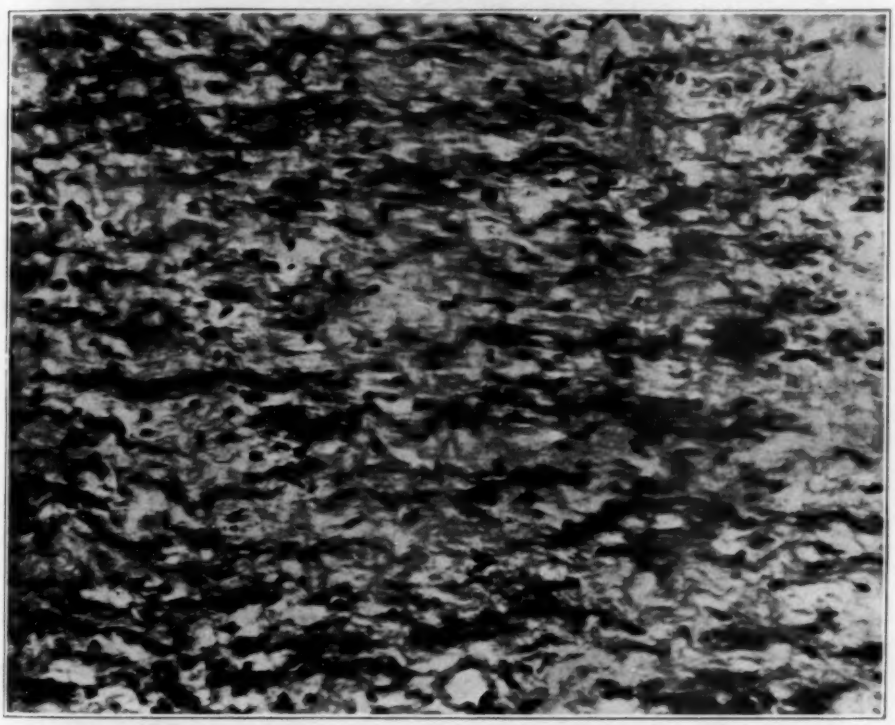

Fig. 2 (case 2).—High power view of the neurofibrillar structure of benign neurofibroma of the pharyngeal wall.

of destruction, the postaural wound was left open for inspection, and it was planned to close it later.

Microscopic Examination of Tissue from the Area of the Right Mastoid.—"A direct smear showed no acid-fast bacilli. We find no neoplasm thus far. The dense cellular infiltrate shows an unusually high component of eosinophils and plasma cells. No tubercles have been found. At present this is our diagnosis: chronic infectious granulation tissue; chronic osteitis and periostitis."

Further Treatment and Course.—Because the convalescence was normal, the postaural wound was closed and on April 6 tonsillectomy and adenoidectomy were done.

At this time I felt that the two most likely possibilities were (1) local tuberculosis with secondary infection and (2) some type of cyst, such as a dermoid cyst, with secondary infection.

On October 7 the child came for observation. The wound had healed well, and the surgically enlarged external auditory meatus had completely closed over with

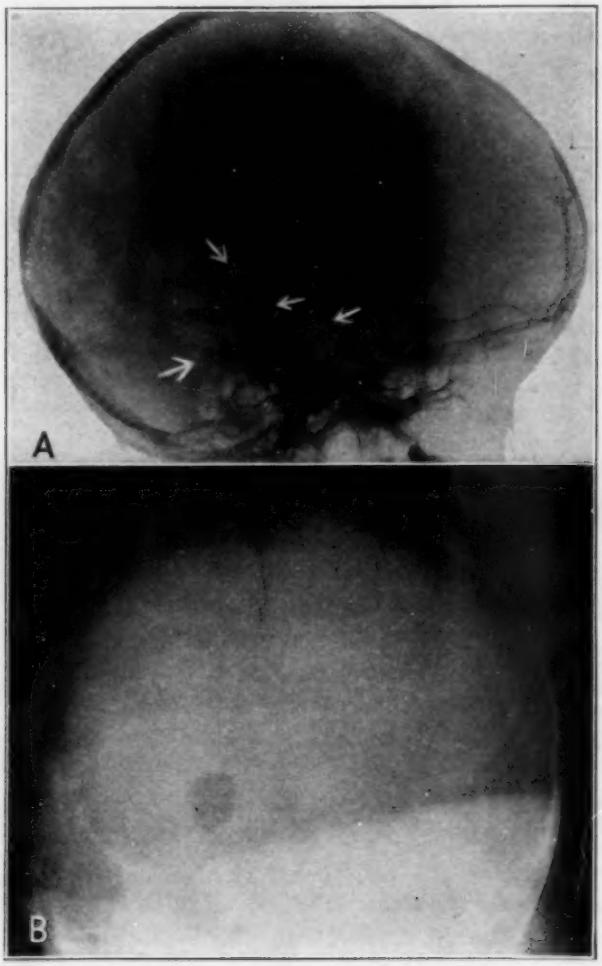

Fig. 3 (case 3).—*A*, lateral roentgenogram of the skull, showing an infected xanthoma involving the right mastoid and destruction anterior to the ear in the squamous portion of the temporal bone. *B*, anteroposterior roentgenogram of the skull. The punched-out area in the occipital bone is characteristic of the destruction caused by xanthomatosis.

a thin atropic skinlike membrane. Inasmuch as there was no perceptible cochlear function, I felt that this was a happy result and left this membrane intact. Roentgen studies of the skull at this time showed normal conditions except for the defect due to operation.

On Sept. 27, 1932 the child was brought for reexamination, because of a swelling about 1 inch (2.5 cm.) in diameter under the scalp in the occipital region. Roentgen study showed a clear punched-out area 2 cm. in diameter in the occipital bone, and this gave me my first clue to the diagnosis of xanthomatosis. On being questioned, the mother stated that the child drank a great deal of water and voided frequently during both day and night. The urine showed a low specific gravity —1.003—but was otherwise normal. Complete roentgen study of the long bones and pelvis disclosed no further pathologic change at this time. The blood count was normal with the exception of a slight secondary anemia. Repeated urinalyses over a period of several weeks showed a high point in specific gravity of 1.004, with occasional recessions to points as low as 1.000. In all examinations the urine failed to show Bence Jones protein. Phenolsulfonphthalein excretion tests indicate normal renal function. The blood cholesterol, determined at several times, varied from 187 to 190 mg. per hundred cubic centimeters.

On Oct. 7, 1933 the child was examined after roentgen therapy. Roentgenograms showed great improvement in the lesion of the skull.

On Feb. 2, 1934, a small area of destruction, which was taken to be an area of xanthoma, was found by roentgen examination in the distal third of the right femur. Since the administration of roentgen therapy this patient has continued to be well up to the time of writing and has had no further marked recurrence of bony lesion. Administration of solution of posterior pituitary was tried in this case but resulted in only slight temporary control of the diabetes insipidus. A low fat diet was tried but seemed to be of little or no value.

Comment.—In my opinion a diagnosis of xanthomatosis was justified because of the presence of the triad of clinical symptoms: (1) defects in the membranous bone, (2) exophthalmos and (3) diabetes insipidus. Although, according to the description given, no foam cells were found in the tissue removed, I feel that the numerous surgical attacks and secondary infection might well account for this Xanthoma ranks cellularly as a benign tumor, but the tumor was so located in this child that it caused complete destruction of the function of the right ear, and so it deserves to be called malignant by position.

In the literature there are many interesting references to xanthomatosis, or Hand-Schüller-Christian disease. All authors agree that pathologically xanthoma is characterized by foam or lipoid cells and the older lesions by cellular reactions and fibrosis.

Pick [7] stated that xanthomatosis is a congenital familial disease on a constitutional basis and that it occurs mostly in Jews and in males. The predilection for members of the Jewish race is probably a coincidence of determining hereditary factors favored by the fact that among Jews marriages between persons who are related by blood are frequent.

Davison [8] stated that there are three possible causes: (1) that the growth is neoplastic or that it is sarcomatous, (2) that it is a neoplasm secondary to trauma and (3) that it is inflammatory.

7. Pick, L.: Niemann-Pick's Disease and Other Forms of So-Called Xanthomatosis, Am. J. M. Sc. **185**:601 (May) 1933.

8. Davison, C.: Xanthomatosis and the Central Nervous System, Arch. Neurol. & Psychiat. **30**:74 (July) 1933.

Reich [9] stated that a xanthoma is a specific granuloma, caused not by micro-organisms but by a disturbance in the stability of the emulsion of cholesterol and fat in the blood.

Druss [10] made an interesting comment with regard to the histologic findings, which is consoling to me in view of the microscopic report on the tissue which was removed in this case. He said

> the foam cells disappear in advanced lesions, and with the leukocytes, lymphocytes, plasma cells, and spindle cells present, the tissue appears like granulation tissue. In advanced lesions the foam cells disappear and the tissue becomes vascular and undergoes fibrosis.

Another report [11] stated that older lesions may consist almost entirely of benign foreign body giant cells, fibrous tissue or even necrotic tissue.

In 1936 Wortis, Wolf and Dyke [12] stated that only 57 cases of xanthomatosis of the Hand-Schüller-Christian type had been reported in the literature, but in 1937 Atkinson [13] listed 103 cases in an excellent review of all the literature to date.

Several cases have been reported in which the mastoid and temporal bones were involved. Several instances of involvement of the external ear, middle ear, mastoid region, labyrinth and inner ear, and also instances of facial paralysis have been reported, All authors agree that roentgen therapy is the treatment of choice. Diet and endocrine therapy have been tried, but their value is doubtful.

Rowland [14] stated that in the healing after roentgen therapy the infiltration in the mastoid region may lead to a deposit of dense bone, with resulting deafness.

Shea [15] reported a case in which there was involvement of the mastoid. The diagnosis was made by roentgen examination, which showed other lesions, and roentgen treatment resulted in recovery. He gave an excellent review of the literature and stated: "Surgery has no place in the treatment of this disease." I heartily concur in this opinion but feel that the circumstances of repeated previous operation with existing secondary infection warranted the procedure in my case.

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9. Reich, S.: Xanthomatosis and Schüller-Christian Syndrome: A Roentgenological and Clinical Study, Canad. M. A. J. **31**:256 (Sept.) 1934.

10. Druss, J. G.: Aural Manifestations of Lipoid Granulomatosis (Hand-Schüller-Christian's Disease), Ann. Otol., Rhin. & Laryng. **45**:693 (Sept.) 1936.

11. Kennedy, R. L. J.: Xanthomatosis: Schüller-Christian's Disease, Proc. Staff Meet., Mayo Clin. **13**:776 (Dec. 7) 1938.

12. Wortis, S. B.; Wolf, A., and Dyke, C. G.: Xanthomatosis and Syndrome of Diabetic Exophthalmic Dysostosis, Am. J. Dis. Child. **51**:353 (Feb.) 1936.

13. Atkinson, F. R. B.: Schüller-Christian's Disease, Brit. J. Child. Dis. **34**: 28 (Jan.-March) 1937.

14. Rowland, R. S.: Schüller-Christian's Disease, Am. J. Roentgenol. **30**:648 (Nov.) 1933.

15. Shea, J. J.: Xanthomatosis, Laryngoscope **48**:589 (Aug.) 1938.

CASE 4.—Mrs. R., 53 years old, came to me on May 18, 1932, complaining of a tender swelling involving the left cheek. There were complete obstruction of both nostrils and great difficulty in swallowing. A constant dull pain was present around the left side of the face. There was a history of eight operations on the left side of the upper jaw during the last eight years since this growth had been present. Other than this her personal history, marital history and family history were essentially noncontributory.

On examination there was a large perforation in the roof of the mouth, involving practically all the hard palate and most of the soft palate. Through this perforation could be seen a new growth involving both antrums and both ethmoids, which had practically destroyed the septum and both nasoantral walls. There was considerable invasion of the soft tissues of the left cheek. One upper tooth remained, the third molar on the right. The ears and throat were essentially normal. There was a foul odor, due to the secondary infection of the tumor mass involving the mouth and nose. A roentgenogram showed destruction which corresponded to the clinical picture. A specimen was taken for biopsy, and the following report was returned:

"The specimen is a growth from the left naris and the floor of the left antrum. It is a neoplastic growth fitting into the diagnosis of recurrent adamantinoma. At places there is a myxomatous stroma infiltrated with the neoplastic cells, which are here flattened and fusiform. Large areas show only growth of the solid type, without cysts or glandlike spaces. The malignancy of the tumor is largely local, as metastases of growths of this type are rare. While the presence of columnar enameloblasts is difficult to make out, the cell type and the cystic and perivascular arrangements are sufficient for a diagnosis of adamantinoma."

On June 8 the left external carotid artery was ligated.

On June 17 a modified Moure operation was done. The left maxilla was almost totally removed. The left orbit was not grossly involved, and it was not disturbed. The remainder of the septum and both nasoantral walls were removed, with wide removal of all the involved tissue on the right. Radium was used after the operation. Dr. W. M. Robey, a local dentist, made an unusual flanged dental appliance and obturator, which closed the large opening through the roof of the mouth and thereby facilitated dressings of the wound and contributed greatly to the comfort of the patient. Before the patient was dismissed from the hospital, a new arrangement of this dental appliance was made, with a complete upper plate of false teeth attached.

She was seen next in September. There was no odor or pain at this time. She could eat well and had gained in weight from 78 pounds (35 Kg.) to 130 pounds (59 Kg.) since she was first seen by me, three months before.

On April 25, 1933, because there was a small recurrence around the inner canthus and a beginning corneal ulcer on the left, and since pathologic report showed that the grade of malignancy was increasing, it was necessary to remove both eyelids and the contents of the left orbit. Again radium was used. Although there was not a great amount of hemorrhage, the patient suffered considerable shock and survived the operation by a narrow margin.

On April 1, 1934 only small areas of recurrence were seen on the margin of the soft palate and the base of the sphenoid bone, and these were destroyed by electrocoagulation.

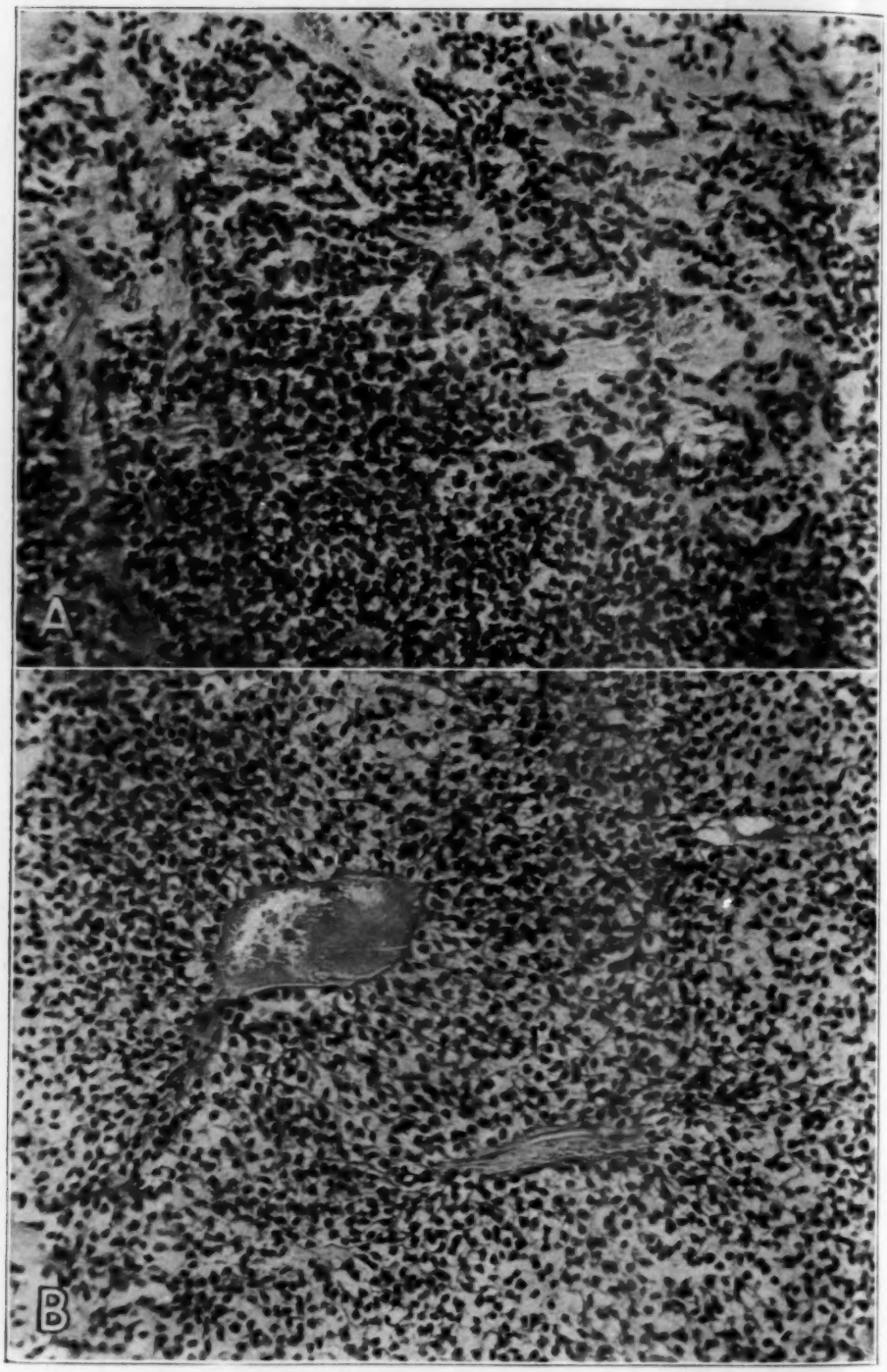

Figure 4

(See legend on opposite page)

I received the report that this patient carried on her usual activities and was well up to a month before the time of her death, which occurred in 1937 and was the result of a secondary intracranial extension of the growth through the ethmoid and sphenoid areas into the base of the brain.

Fig 5 (case 4).—The patient after the second operation for recurrent adamantinoma.

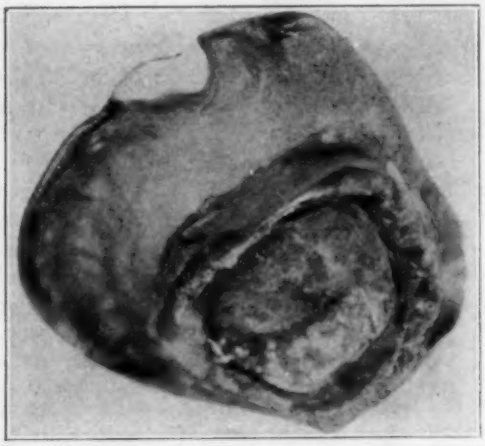

Fig. 6 (case 4).—The dental appliance and obturator worn by the patient.

EXPLANATION OF FIGURE 4

Fig. 4 (case 4).—Recurrent adamantinoma. *A*, a high power view of the periphery of the growth, showing atypical enameloblasts in a loose mucinous stroma. A tendency to the formation of small cystic spaces is seen. *B*, a high power view of the area of the solid growth of atypical enameloblasts.

Comment.—Adamantinoma is usually considered a benign tumor. Undoubtedly, this tumor in its incipient stage was benign in both cellular structure and location, but by succeeding recurrences after inadequate surgical intervention and by gradual extension into the orbital and ethmoid regions, malignancy by position was attained. Adamantinoma is not a common tumor and is conceded by most authors to be of much more frequent occurrence in the lower jaw than in the upper jaw. However, this is not the experience of Hertzler,[16] who stated that he has encountered the tumor much more frequently in the upper jaw. Hertzler also classified adamantinoma with mixed tumors. He expressed the belief that they are a great deal alike in many respects.

Davis [17] stated that adamantinomas probably arise from supernumerary remnants of the dental anlage.

Major, Bell and DeWaters [18] stated that adamantine tumors are more apt to recur and more apt to attain a high degree of malignancy in the upper jaw than in the lower jaw. They also stated that metastasis is rare but that it is occasionally reported.

Ivy and Curtis,[19] Ghosh,[20] Kau and Wong [21] and others have agreed that conservative surgical procedure has no place in the treatment of adamantine tumors and that radical removal should be done, to lessen the chance of recurrence. Roentgen rays and radium should be used only in conjunction with surgical intervention.

CASE 5.—T. B., a 12 year old mountain boy, came to me for examination on May 22, 1930. He had had swelling and protrusion of the left eye for the last six months. There was marked nasal obstruction, and there had been loss of weight due to difficulty in swallowing.

On transillumination the left antrum and the left frontal sinus appeared dull. A large, grayish pink mass filled the left nostril and extended into the nasopharynx. Roentgen examination showed bony destruction suggestive of a new growth involving the nasofrontal region on the left and filling the left antrum, with distortion of the orbital structures on the left. The following report was received from the department of pathology at the Johns Hopkins University: "Myxoma. It is a rare tumor, usually benign, but may show signs of malignancy and may also recur."

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16. Hertzler, A. E.: Adamantinomas, in Surgical Pathology of the Mouth and Jaws, Philadelphia, J. B. Lippincott Company, 1938, p. 166.

17. Davis, A. H.: Adamantinoma, J. Oklahoma M. A. **29**:1 (Jan.) 1936.

18. Major, S. G.; Bell, J. R., and DeWaters, R. S.: Adamantine Tumors of the Jaws, Surg., Gynec. & Obst. **59**:876 (Dec.) 1934.

19. Ivy, R. H., and Curtis, L.: Adamantinoma of the Jaw, Ann. Surg. **105**: 125 (Jan.) 1937.

20. Ghosh, L. S.: Adamantinoma of the Upper Jaw, Am. J. Path. **10**:773 (Nov.) 1934.

21. Kau, L. S., and Wong, A. S.: Adamantinoma of the Upper Jaw, Chinese M. J. **50**:37 (Jan.) 1936.

The following report came from Dr. A. S. Warthin, of the department of pathology of the University of Michigan: "Myxochondroma. While not very cellular, it should be treated as sarcoma. It will not metastasize in its present degree of cellularity but may become more cellular with successive recurrences."

On May 23, with the patient under general anesthesia, a modified Moure operation was done. The growth filled the left antrum, ethmoid sinus and frontal sinus and was adherent to the septum and turbinates. I feel that its origin was most likely the left ethmoid sinus of the frontal sinus. In addition to the tumor tissue, the bone in the region of the left frontal sinus, the ethmoid sinus and the nasoantral

Fig. 7 (case 5).—Myxochondroma. *A*, high power view of a typical area, showing embryonic chondroblasts in a mucinous intercellular substance.

wall was removed. Radium was used after the operation. The lower part of the incision in the cheek was left open, so that the involved area of the sinuses might be kept under observation for possible recurrence of the growth.

In June 1932, the boy had gained weight, and his general condition was excellent. Inasmuch as there was no sign of recurrence after two years, the opening in the cheek was closed. The patient has reported occasionally to the time of writing, September 1939, and there has been no recurrence of his tumor.

Comment.—I feel that this tumor should be classed as malignant by position. Although it is designated histologically here as "potentially

malignant and with possibility of recurrence," the cellular structure is usually that of a relatively benign tumor.

I am able to find but one direct reference to myxochondroma in the literature at my disposal. McAuliffe [22] reported a myxochondroma arising from the region of the right inferior turbinate and removed a tumor the size of a lemon from a boy 8 months old. In the microscopic examination of this tumor he found no evidence of malignant change, but he kept the patient under constant observation for possible recurrence or malignant change. He stated that myxochondroma is very rare and that it occurs in any region but least frequently in the nose. I feel that his excellent report of this type of tumor, the size of a lemon, in a boy 8 months of age, is more unusual than my report. In his case radiation was not used.

SUMMARY

Five relatively benign tumors are considered in this paper:

1. An ependymal cyst of the choroid plexus (in association with chronic otorrhea and chronic mastoiditis, which necessitated surgical intervention). This tumor caused the death of the patient by obstructing the flow of cerebral spinal fluid.

2. A large neurofibroma of the lateral pharyngeal wall. The surgical removal of this tumor was accompanied by serious risk, because of the proximity and necessary exposure of the great vessels of the neck.

3. An infected xanthoma involving the petrous portion of the temporal bone. Surgical removal of the tumor was necessary. There was a resulting loss of cochlear and vestibular function, as well as a threat of intracranial infection.

4. A recurrent adamantinoma of the upper jaw. The malignancy of the tumor increased at each recurrence. After a period of years, the adamantinoma gradually extended through the skull to the base of the brain and caused death.

5. A myxochondroma of the nasofrontal region so large as to distort the orbit greatly and disfigure the patient. Extension of the growth through the posterior choana to the pharynx caused emaciation, due to difficulty in swallowing.

These tumors in different locations and offering a great variety of cellular construction are grouped together here because by virtue of their vital position they have a common factor of position malignancy.

Dr. L. C. Todd, Professional Building, Charlotte, N. C., prepared the pathologic reports and aided in obtaining the photomicrographs.

22. McAuliffe, G. W.: Myxochondroma in Nose of Child Eight Months Old: Report of Case, Laryngoscope **48**:206 (March) 1938.

MASSIVE DOSES OF VITAMINS A AND D IN THE PREVENTION OF THE COMMON COLD

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During their various studies of the clinical use of massive doses of vitamin D, Reed and associates [1] noted that many patients receiving large doses of vitamin A and vitamin D claimed that both the incidence and the severity of their colds were appreciably reduced. No accurate statistics were kept, but it appeared to them that only those subjects receiving both vitamins were thus benefited.

This observation was not surprising since many investigators have noted great reduction in the incidence of colds and respiratory infections through the use of fairly liberal quantities of cod liver oil. Holmes and his colleagues,[2] for example, reviewed the subject and showed a reduction of about two thirds in the average "lost time" of industrial workers due to colds and respiratory diseases when cod liver oil was furnished. In general Holmes felt that the reduction in colds was due to the vitamin A content of the cod liver oil, although no proof of this could be offered. The doses used by Reed and associates, however, were many times larger than those used by previous workers, and the subjects could not be said to be deficient in vitamins A or D. Whatever effects appeared, therefore, were almost certainly due to some cause other than correction of vitamin deficiency.

In view of these facts, it was of interest to study the effects of vitamins A and D in large doses, both singly and combined, on a group of subjects. The purpose of this study was to determine, first, whether any amelioration as regards the incidence and severity of colds was achieved; second, whether one or the other of the vitamins was alone responsible for the effects or whether both were necessary.

From the Department of Otolaryngology of the University of Illinois College of Medicine.

1. Reed, C. I.: Personal communication to the author. Reed, C. I.; Struck, H. C., and Steck, I. E.: Vitamin D, Chicago, University of Chicago Press, to be published.

2. Holmes, A. D.; Pigott, M. G.; Sawyer, W. A., and Comstock, L.: The Influence of Body Weight and the Administration of Cod Liver Oil on Industrial Absenteeism, J. Am. Dietet. A. **10**:208, 1934; The Value of Cod Liver Oil for Building Bodily Resistance of Industrial Workers, Oil and Soap **12**:86, 1935; Cod Liver Oil: A Five Year Study of Its Value for Reducing Industrial Absenteeism Caused by Colds and Respiratory Diseases, Indust. Med. **5**:359, 1936.

PROCEDURE

Fifty-four patients were studied during the three winters from 1934 to 1937. Thirty-two of these subjects were studied for all three winters, 18 for two years and 4 for only one winter. Thirty-four of the subjects were men, and the rest were women. Their ages ranged from 7 to 49 years, with 14 of the subjects 16 years old or younger. All the patients belonged to the group called "chronic or frequent cold sufferers" (Spiesman and Arnold [3]) and had previous records of five to seven colds each winter for several years previous to the present study. These colds were accompanied by fever and lasted for periods of from seven to fourteen days. Many patients required rest in bed. All patients chosen for this study were free from pathologic conditions as determined by close examination, particularly of the respiratory tract and nasal sinuses. It was felt that this precaution would insure results unobscured by extraneous factors.

Without regard to age or sex the subjects were divided into 3 groups as follows: Group 1 received only vitamin A; group 2 received only vitamin D, and group 3 received both vitamins A and D. There were 7 patients in group 1; 7 patients in group 2 and 40 patients in group 3. The explanation for the small number of subjects in groups 1 and 2 is that it was difficult to persuade the subjects in these groups, who derived no benefit from the use of vitamin A or D alone, to continue the treatment over longer periods.

The vitamin A used was in two forms: a solution of carotene in cottonseed oil [4] containing 7,000 international units of vitamin A per gram and a fish oil concentrate of vitamin A [5] containing 40,000 international units of this vitamin per cubic centimeter, stated to be free of vitamin D. The vitamin D [6] used was in the form of a solution of calciferol in oil, containing approximately 920,000 international units per cubic centimeter.

Treatment was started in all cases about September 1 and continued throughout the winter and spring until about June 1. The initial dose of vitamin A as carotene solution for subjects in group 1 was approximately 900 units per day. For those receiving the fish oil concentrate the dose was approximately 9,000 units per day, taken in two doses with meals. In group 2, the initial dose of vitamin D was 120,000 units per day. In group 3 the subjects started with 9,000 units of A and 120,000 units of D daily. In all cases, the dose was increased by gradual stages as follows: For patients taking the carotene solution the dose was increased by approximately 400 units every third day up to 7,000 units daily. For those receiving the fish oil concentrate the dose was increased by about 2,500 units every third day until the maximum dose of 40,000 units was reached. For subjects receiving vitamin D the dose was increased by 60,000 units every third day until a maximum of 300,000 units was reached. In a similar manner the doses of vitamin D, after varying periods, were gradually reduced to the initial dose. Children under 12 years of age received half these amounts.

All the subjects were examined weekly or semimonthly throughout the season, and records were kept of the number and severity of colds experienced by them.

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3. Spiesman, I. G., and Arnold, L.: Host Susceptibility to Common Colds, Am. J. Digest. Dis. & Nutrition **4**:438, 1937.

4. Furnished by the S. M. A. Corporation.

5. Furnished by the Glaxo Laboratories.

6. Furnished by Mead Johnson & Company.

RESULTS

The table summarizes the results of the study. It shows that none of the subjects who received either vitamin A or vitamin D alone derived any benefit from the treatment; that is to say, they experienced

Summary of Results

Subject	Age, Years	Sex	Vitamins	Result
1	5	F	A*	
2	41	M	A	
3	19	M	A*	
4	23	F	A	
5	25	F	A*	
6	31	F	A	
7	39	M	A	
8	19	F	D	No benefit
9	24	M	D	
10	37	M	D	
11	18	F	D	
12	47	M	D	
13	32	M	D	
14	25	F	D	
15	7	M	A and D	
16	10	M	A and D	
17	19	F	A and D	Free of colds during second and third years
18	23	M	A and D	
19	31	M	A and D	
20	33	F	A and D	
21	11	M	A and D	
22	28	F	A and D	
23	49	M	A and D	
24	41	F	A and D	
25	27	F	A and D	
26	18	M	A and D	
27	29	M	A and D	
28	16	M	A and D	
29	9	M	A and D	
30	48	M	A and D	
31	37	F	A and D	
32	11	F	A and D	
33	22	M	A and D	Colds reduced in number and severity
34	18	M	A and D	
35	30	M	A and D	
36	14	M	A and D	
37	12	M	A and D	
38	32	F	A and D	
39	25	F	A and D	
40	19	M	A and D	
41	42	M	A and D	
42	12	M	A and D	
43	27	M	A and D	
44	14	M	A and D	
45	29	M	A and D	
46	47	F	A and D	
47	38	F	A and D	
48	31	M	A and D	
49	27	M	A and D	
50	18	M	A and D	No benefit
51	14	M	A and D	
52	10	M	A and D	
53	9	F	A and D	
54	11	F	A and D	

* Vitamin A was given in the form of carotene solution; in all other instances this vitamin was given in the form of a fish oil concentrate.

just as many colds (five to seven) and the colds were just as severe as those of the seasons in which they did not take the vitamins. On the other hand, of the patients who received both vitamins, 6 (15 per cent) were completely free of colds during the second two years of

the treatment, and during the first winter experienced a marked reduction in number (three or less) as well as a definite decrease in severity. In the group which received both vitamins, 8 (20 per cent) received no benefit from the treatment, while 26 (65 per cent) experienced a reduction in both number and severity. This is to say that the number of colds per year dropped to three and that the average duration was five days, with but little elevation of temperature. When these conditions were not fulfilled, the subject was classified as having received no benefit from the treatment.

It was apparent that the combination of vitamins A and D reduced the incidence and severity of colds in the majority of susceptible persons. This is not to say that ingestion of vitamins in the doses mentioned is to be considered as curative or completely preventive. The results point only to the striking difference between those subjects treated with both vitamins and those treated with only one.

Symptoms of toxicity due to one or another of the vitamin preparations developed in a few of the subjects. All 3 of the patients who received the carotene solution had symptoms of toxicity after taking 7,000 units daily for periods of from four to fourteen weeks. The symptoms were carotenemia of high degree as evidenced by yellow coloration of the skin and eyes, anorexia, loss of weight and a general feeling of malaise. One patient (no. 6) receiving only vitamin A as fish oil concentrate showed similar symptoms except that hypercarotenemia was absent. Two patients (nos. 10 and 12) receiving only vitamin D also exhibited signs of toxicity, the symptoms being headache, polyuria, nausea and anorexia. This toxicity occurred six and eleven weeks, respectively, after treatment with 300,000 units daily. No subject in group 3, receiving both vitamins A and D, displayed any symptoms of toxicity at any time during the study.

In every case of toxicity the vitamin intake was stopped immediately, and a 10 per cent solution of calcium levulinate was given intravenously in doses of 10 to 20 cc. daily for five days, or a 10 per cent solution of calcium chloride was given by mouth, 1 tablespoonful (15 cc.) four times daily until symptoms vanished. In all cases the symptoms disappeared fairly rapidly under these conditions, requiring about four to ten days to vanish completely. No other injuries were apparent, and after several weeks of rest the patients were again given the vitamins but in doses only half as great as previously.

SUMMARY

Vitamins A and D in massive doses did not produce immunity to the common cold when given separately.

When massive doses of vitamins A and D were given together, 80 per cent of the subjects showed a significant reduction in both the

number and the severity of common colds. The number of colds per year dropped to three, and the average duration was five days, with but little elevation in temperature.

It must not be assumed here that the use of vitamins in the treatment of the common infectious cold is offered as a panacea for colds. Average susceptibility must be taken seriously into account, as well as the emotional state of the subject (Spiesman [7]) as affected by an innately unstable vasomotor mechanism.

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7. Spiesman, I. G.: Vasomotor Responses of the Mucosa of the Upper Respiratory Tract to Thermal Stimuli, Am. J. Physiol. **115**:181, 1936.

MANAGEMENT OF SINUSITIS IN CASES OF BRONCHIECTASIS

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Sinusitis is a frequent complication of bronchiectasis. Its prevalence varies with the experience of different observers, but I think all agree that it occurs sufficiently often to constitute an essential part of the picture of bronchiectasis. The purpose of this paper is to present some observations on the surgical management of cases in which operation is indicated and to outline briefly the points to be considered in other cases in which operation is inadvisable.

Recently, I had an opportunity to survey 150 cases of bronchiectasis at the pulmonary clinic of the Massachusetts General Hospital and to evaluate the effect of surgical intervention both on the symptoms and on the economic effectiveness of the patients. The following observations are drawn from a study of these cases. Over 90 per cent of the patients had sinusitis in some degree.

Patients with sinusitis may be classified according to the severity of this complication and according to the more general division of age, susceptibility to infection of the upper respiratory tract, extent of the pulmonary process and ability to withstand operative procedures. These considerations make it necessary to individualize each case. This means not only that otolaryngologists should have consultations and close cooperation with the general medical service but also that within the field of rhinology they should apply measures which are indicated for the individual patient.

The local problem in the nose and sinuses remains for the most part the same as in uncomplicated cases, and here I follow the principles described by Porter [1] for the treatment of acute and chronic sinusitis. The pulmonary element adds to this picture the factors of reinfection of the chest from above and the additional load of infection the patient must bear. In general, the aim of any treatment of the sinuses in these cases is the drainage of infected secretions, the removal of diseased tissue or the correction of factors which cause tissue to be infected. Of course, it is not possible to remove the bronchiectatic cavity by sinus

Read at the Forty-Fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Cleveland, Oct. 9, 1940.

1. Porter, C. T.: Sinusitis: The Present Rationale of Treatment, Ann. Otol., Rhin. & Laryng. **48**:769-774 (Sept.) 1939.

surgery. However, it can be relieved of whatever infection may reach it from the sinuses by any of the routes described by Fenton and Larsell.[2] The removal of one focus of infection can lighten the total load so that the patient will have more strength to combat the thoracic disease.

In the series of cases I studied,[3] I found that bronchiectasis had developed in one third of the patients before the age of 10. This group presented some factors which set it apart from the older patients. They attributed the bronchiectasis to essentially pulmonary diseases rather than to sinusitis. They gave a history of pneumonia, pleurisy, whooping cough or bronchitis as the direct precursor of the disease. However, these patients seemed extremely susceptible to colds, and sinusitis developed in many of them either at the time of the original infection or afterward. They had low resistance to all infections. As they could not stand operations as well as children without bronchiectasis, extensive sinus operation was not indicated.

The nasal problem seemed to be a mechanical one; they did not seem able to keep the nasal passages free and clear from secretion. Their turbinates were swollen and often bathed in tenacious mucopus; this they endured without attempting to get rid of it. Some of them did not know how to blow the nose, and for many even forceful blowing did not bring relief. This retention of pus augured more chronic infection of the sinuses.

Operations when indicated for children with bronchiectasis should aim at drainage. Intranasal window operations on the antrum, removal of anterior tips of the middle turbinate and adenoidectomy are the operations usually performed. After operation, suction or lavage of the nose helps to remove what secretion the patient cannot blow out. In cases in which operation was contraindicated, I have used suction with considerable benefit to some of the patients. Most of the others, even those in whom the objective improvement was only partial, experienced subjective relief which lasted from three to four days. In any event this treatment seems to be a valuable adjunct to other procedures in the management of the disease.

Finally, in treating patients below the age of 10, great attention should be paid to the general hygiene. As a rule, it is left for the pediatrician to decide questions of diet, management of nursing, in the home, the sanatorium or the hospital, and the desirability of immunization with bacterial vaccines. He should also watch carefully the environmental factors and, if it is possible, consider a change to a better climate.

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2. Larsell, O., and Fenton, R. A.: Lymphatic Pathways from the Nose, Arch. Otolaryng. **24**:696-713 (Dec.) 1936.

3. Goodale, R. L.: An Analysis of Seventy-Five Cases of Bronchiectasis from the Viewpoint of Sinus Infection, Ann. Otol., Rhin. & Laryng. **47**:347-349 (June) 1938.

There is another group which must be considered conservatively, patients who have endured bronchiestasis and sinusitis well past middle age. In many the expectancy for many more years of life is slight. Palliation of symptoms or at most minor surgical procedures will give these patients the best results.

In the third group, young adults and adolescents, when the ability of the patient to stand operation is sufficient, radical surgery can be considered. Unfortunately, in this group there are some with such an overwhelming complex of diseases that conservative measures are all that can be attempted. For those people who can stand it, surgical intervention offers the greatest relief. Some of these patients may not need more than correction of those factors which contribute to reinfection of the sinuses. They may not actually have a chronic disease, but they are nevertheless subject to repeated attacks of acute sinusitis. Here again, submucous resection, removal of middle turbinates, intranasal operation on the antrum and tonsillectomy may be the only surgical procedures necessary.

It is remarkable that there is a high incidence of severe chronic sinusitis in this age group. Frequently, roentgenologic examination shows not only marked thickening and retained secretion but also bone reaction. In this severe form the disease may be of the silent type, and the patient may not be aware of the extensive nature of his condition. In fact, at times the intranasal examination will fail to indicate how severe the infection really is. For this reason, roentgenologic examination of sinuses is absolutely essential, and in many cases more than one series of films may be necessary to evaluate the severity of the disease.

I have been impressed by the number of patients who suffer from pansinusitis. In fact, the ethmoid cells seem to be as frequently infected as the antrums, and the infection of both the antrum and the ethmoid labyrinth is the more common history. The sphenoid sinuses also seem to be more often infected than was previously thought. I am inclined to give the ethmoid cells more and more consideration as I see the results of complete and incomplete operations on the sinuses. I have not observed the dryness and crust formation which many fear result from complete ethmoidectomy. In my opinion, purulent crusts are more apt to occur when some infected cavity is still present. The best results have been observed in those cases in which the operation has been thorough and the recuperative powers of the patient were good from the start.

I shall not discuss the technics of operating except to say that for radical operations on the antrum I favor an approach through the canine fossa, and for operations on the ethmoid and sphenoid sinuses, a combination of the intranasal and the transantral route. An external orbital approach may be preferred sometimes, especially if the frontal sinus is involved.

In general, the points to be considered in deciding for radical operation are the same as in cases without thoracic complications. However, as I have said, the latter type of complication adds new factors, namely, the danger of reinfection of the chest from above and the inability of the patient to withstand both the injury of multiple infections and operative procedures. Some patients are generally so infirm that they cannot stand much operating at one time. The age of the patient, as I have pointed out, is also to be considered.

There is likewise a seasonal factor which applies especially to New England. The danger of epidemic infections during the autumn, winter and early spring is a real one for patients with pulmonary disease. They are extremely liable to repeated attacks of pneumonia, and minor operations, not to mention major ones, will frequently tip the scales against them. For this reason the thoracic service of the Massachusetts General Hospital has decided to perform lobectomies preferably during the late spring and summer months, and I feel that is a good example to follow in operating on the sinuses of the same patients. Then, too, during the summer there are more weeks after operation in which there is less danger of infection. This allows a healthy repair of the operative field. I wish to emphasize especially the undesirability of operating whenever there is an epidemic of head colds. Likewise, there should be no remnant of an acute infection latent in the sinuses at the time of operation. The pulmonary infection should also be as quiescent as possible. Routine postural drainage is advocated for bronchiectatic patients; it is advisable to have the chest drained out in this way as completely as possible before operation, as it will be necessary for the patient to be quiet for one or two days after the operation. In one or two instances, postural drainage has started postoperative bleeding. On the whole, sedatives which reduce cough should not be used, as they tend to allow secretions to accumulate in the chest. If the patient is in a ward, he should be isolated whenever acute infection breaks out among the other patients.

To return to the operation, I have already mentioned that it is desirable to do as complete an operation as the general condition of the patient will allow. If possible, this operation should be completed in one stage. When much has to be done or when the patient's general condition makes it necessary, the operation must be divided into several stages. It is better not to overoperate at any one time. For instance, at the first operation it may be possible to correct only a badly deviated septum. At the second operation, one may be able only to clean up the antrum or the ethmoid labyrinth on one side, leaving the opposite side for still a third operation. In such a situation, I think it is best not to have the operations come close together. Give the patient enough time between operations to recover from whatever postoperative reaction he has experienced.

One other point is extremely important, and that is to have adequate bacteriologic studies made before operation and also from the pus obtained at the time of operation, to anticipate any necessity for chemotherapy.

In regard to anesthesia, I have always preferred the local type for fear of lighting up the pulmonary infections with ether, although recently the medical service and thoracic surgeons have seen no contraindications to ether. In fact, lobectomy itself is frequently done with the patient under general anesthesia.

I should like to bring out one other point. The infected sinuses and the infected chest can be considered as two separate foci of infection, which together constitute the patient's disability. The removal of infection from one without the removal of infection from the other is an incomplete operative procedure. Just as complete is better than halfway operating, so the best results follow lobectomy plus a thorough sinus operation.

Finally, the only way to insure success in operating is to follow the case for both observation and treatment of later conditions that threaten a return of sinusitis, for example, recurrent infections of the upper respiratory tract. This follow-up and after-care are as much a part of the management as the preoperative consideration of the case and the operation itself.

SOME EXPERIENCES WITH SINUSITIS IN SWIMMERS

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Although the dictum "Avoid bone work in the treatment of acute sinusitis" must be modified in some instances, it should be diligently observed by every rhinologist. It has been recognized for years that osteomyelitis of the skull, brain abscess and meningitis have developed subsequent to inopportune surgical attacks on acutely infected paranasal sinuses. Hence recent contributions to otologic literature advocating early surgical drainage of the frontal sinuses in cases of acute fulminating infection, such as that which follows swimming and diving, have prompted this report.

In this discussion I shall defend the conservative management of such sinusitis and attempt to present a rationale of surgical treatment based on the pathogenesis of intracranial complications. I shall also present a brief summary of data on 13 patients with sinusitis after swimming admitted to the University Hospital in the past ten years (1931 to 1940 inclusive).

Although many otolaryngologists agree that severe acute paranasal sinusitis has followed swimming in some instances, this does not mean that intracranial complications develop in all cases of sinusitis after swimming, even though the condition early reveals characteristics suggesting a fulminating process. Simpson [1] in discussing fulminating sinusitis warned that "all swelling and edema of the frontal region is not osteomyelitis."

A cursory review of recent literature reveals no unanimity about the most efficacious method of treatment. Likewise, little specific information is gained in respect to the incidence and reason for development of acute fulminating sinusitis after swimming.

Read at the Middle Section Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, Jan. 27, 1941.

1. Simpson, W. L.: Surgical Treatment and Its Complications in Cases of Acute Sinusitis, Arch. Otolaryng. **32**:250-255 (Aug.) 1940.

McNally [2] commented on the relative rarity of acute fulminating frontal sinusitis, although Faulkner [3] in discussing McNally's paper mentioned that he had seen 3 or 4 cases a year for several years.

The mechanism of the production of this disease has been the subject of interesting speculation. It has been thought for many years that contamination of water in lakes and pools was responsible for the sinus and ear infections associated with bathing. Hasty [4] stated that the "water in the pools during the time of swimming represents the combined washings, so to speak, of the nasal mucous membranes of every swimmer." Mezz [5] mentioned a personal communication from Samuel Skillern to the effect that "one swimmer with a sinus infection can easily pollute the pool water, which may be taken into the nostrils of his fellows in a concentrated form." Reinking [6] expressed the view that in ocean bathing the force of the waves may carry decayed animal and vegetable matter into the nose and sinuses and that this may be responsible for an acute infectious process.

Taylor [7] concluded that contamination of water was but one possible factor causing ear and sinus infections subsequent to swimming. He stressed man's lack of adaptation to an aquatic environment and demonstrated that there is a considerable reduction in body temperature from swimming in an indoor pool (73 F.) for forty-five minutes; he related this to the lowering of body resistance to infection. Later Taylor [8] with Dyrenforth emphasized the fact that chilling of the body surfaces causes constriction of the blood vessels of the periphery and that this is associated with the constriction of the blood vessels of the nasal mucous membrane. He suggested that prolonged local ischemia of the nasal mucous membrane reduces local resistance and favors infection.

Hill [9] studied the cases of 1,235 students of summer camps in Maine and concluded that the type of swimming or the bacterial content of

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2. McNally, W. J.: Some Considerations Concerning Acute Frontal Sinusitis, Tr. Am. Laryng. A. **56**:97-125, 1934.

3. Faulkner, E. R., in discussion on McNally.[2]

4. Hasty, F. E.: Paranasal Sinus Infection and Swimming, J. A. M. A. **89**:507-509 (Aug. 13) 1927; cited by Mezz.[5]

5. Mezz, D.: Otorhinologic Sequelae of Swimming: Analysis of Present Concepts and New Method of Prevention, Laryngoscope **50**:479-495 (May) 1940.

6. Reinking, F.: Infecciones de nariz y oidos por baños de mar, An. Soc. mex. de oftal. y oto-rhino-laring. **11**:127-128 (Oct.-Dec.) 1936.

7. Taylor, H. M.: Otitis and Sinusitis in the Swimmer, J. A. M. A. **113**: 891-894 (Sept. 2) 1939.

8. Taylor, H. M., and Dyrenforth, L. Y.: Chilling of Body Surfaces: Its Relationship to Aural and Sinus Infections, J. A. M. A. **111**:1744-1747 (Nov. 5) 1938.

9. Hill, F. T.: Otologic Complications from Swimming in Summer Camps: An Attempt at Prevention, Tr. Am. Otol. Soc. **19**:110-126, 1929.

the water did not seem to be important factors in the production of sinusitis or otitis media. He expressed the opinion that the acute head cold was the greatest menace to the swimmer and that it was necessary to restrict the aquatic activities of persons with purulent nasal discharge, a history of sinus disease or asthma and a significant amount of adenoid tissue. Harris in discussing Hill's paper stated that "where there is a latent sinusitis, this may be lighted up by a good deal of bathing." Behrens [10] in discussing the causes of osteomyelitis of the skull mentioned the possibility of inciting chronic sinusitis to dangerous activity by the lowered resistance from chilling that occurs in swimming. McNally [2] noted that in none of his 4 reported cases of acute fulminating frontal sinusitis occurring during the summer months had there been intranasal evidence of chronic sinus disease.

Information gained from current literature regarding the relative frequency of cranial, as compared with intracranial, complications of acute fulminating sinusitis is not enlightening. Skillern [11] stated that "acute frontal sinus infections, especially swimming cases, rapidly develop an acute osteomyelitis." Bucy and Haverfield [12] expressed the opinion that "the most common complications of acute frontal sinusitis, particularly that which follows swimming, are abscess of the frontal lobe and osteomyelitis of the frontal bones." Courville and Rosenvold [13] maintained that intracranial complications of frontal sinus infections are relatively rare.

The method of spread of infection from the frontal sinuses to the diploic structure of the calvarium and to the intracranial contents has been the subject of illuminating research in recent years. Extension of infection by continuity and contiguity of tissue has long been recognized. The importance of the venous channels in the diploe and of the emissary veins connecting the mucous membrane of the frontal sinus with the dural veins was emphasized by Furstenberg [14] and Mosher.[15]

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10. Behrens, H. C.: Osteomyelitis of the Skull of Otitic and Paranasal Sinus Origin, Arch. Otolaryng. **25**:272-304 (March) 1937.

11. Skillern, S. R.: Osteomyelitic Invasion of the Frontal Bone Following Frontal Sinus Disease, Ann. Otol., Rhin. & Laryng. **48**:392-411 (June) 1939.

12. Bucy, P. C., and Haverfield, W. T.: Cranial and Intracranial Complications of Acute Frontal Sinusitis, J. A. M. A. **115**:983-991 (Sept. 21) 1940.

13. Courville, C. B., and Rosenvold, L. K.: Intracranial Complications of Infections of Nasal Cavities and Accessory Sinuses, Arch. Otolaryng. **27**:692-731 (June) 1938.

14. Furstenberg, A. C.: Osteomyelitis of the Skull: The Osteogenetic Process in the Repair of Cranial Defects, Ann. Otol., Rhin. & Laryng. **40**:996-1012 (Dec.) 1931; The Treatment of Acute Nasal Accessory Sinus Disease, ibid. **47**: 902-909 (Dec.) 1938.

15. Mosher, H. P., and Judd, D. K.: An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis, Laryngoscope **43**: 153-212 (March) 1933.

Woodward [16] said that in acute infections of the frontal sinus the ostium is occluded, and pus forms under pressure, which obstructs the blood flow in the mucous membrane, producing necrosis and resulting in thrombosis of the perforating veins. Since it is unlikely that the pressure of pus in an abscess cavity exceeds the pressure of the blood in the neighboring vascular system, it is difficult to accept this theory. Kramer [17] in discussing the intracranial pathways of infection from suppurative processes in the sphenoid and ethmoid sinuses stated that in most of his cases of osteomyelitis he found chronic submucosal abscesses at the primary sites of infection. Because of these observations in the sphenoid and ethmoid sinuses, which were not obvious at operation, he maintained that it would be advisable in cases of meningeal irritation to strip the mucosa from the sphenoid sinuses rather than merely institute adequate drainage. No analogous statement has been found in the literature dealing with frontal sinusitis, although similar conditions might well develop in this area.

There are many references to the bacteriologic characteristics of acute fulminating frontal sinusitis, such as those by Adson and Hempstead,[18] Imperatori [19] and others, which indicate that these processes are of staphylococcic origin.

The general management of acute fulminating sinusitis varies greatly in different clinics. This lack of uniformity in the treatment of a potentially serious acute infectious process tends to prove that the problem of this disease has evaded final solution. Although many clinicians have considered the condition as one for surgical intervention, the type of operation and the best time for its performance have not been uniformly decided.

Skillern [11] expressed the opinion that osteomyelitis of the calvarium after a sinus operation is due in many instances to an inadequate original operation. He inclined to conservatism in the treatment of acute frontal sinus disease until the acute general reactive symptoms have subsided, provided intranasal drainage can be established without incision. He stated that a small trephine opening for drainage of acute empyema of the frontal sinus is not good surgical procedure and may be followed

16. Woodward, F. D.: Osteomyelitis of the Skull, J. A. M. A. **95**:927-930 (Sept. 27) 1930; cited by Adson and Hempstead.[18]

17. Kramer, R., and Som, M. L.: Intracranial Pathways of Infection from Diseases of the Sphenoid and Ethmoid Sinuses, Arch. Otolaryng. **32**:744-770 (Oct.) 1940.

18. Adson, A. W., and Hempstead, B. E.: Osteomyelitis of Frontal Bone Resulting from Extension of Suppuration of Frontal Sinus, Arch. Otolaryng. **25**: 363-372 (April) 1937.

19. Imperatori, C. J.: Symposium on Bacterial Meningitis: III. Differential Diagnosis of Suppurative Meningitis Caused by Paranasal Sinus Disease, with Some Suggested Prophylactic Measures, Laryngoscope **47**:306-310 (April) 1937.

by an osteomyelitic extension. Since he presupposed osteomyelitis in cases of acute frontal sinusitis that demand operation, he advocated thorough curettage of all softened bone and hemorrhagic osteitic matter. Adson and Hempstead [18] insisted that early drainage will prevent osteomyelitis and brain abscess and stated that the infection may involve the diploic veins within forty-eight hours. They agreed, however, that the treatment of acute suppurative sinusitis should be medical during the acute stage but said that it is occasionally necessary to drain the frontal sinus externally at its lower inner angle if severe pain is not relieved. They did not consider craniotomy advisable unless there was evidence of extension into the frontal lobe of the brain. They admitted that infection occasionally develops so rapidly that there is evidence of extension into the cranium before there has been an opportunity to institute surgical drainage of the sinus.

Williams and Heilman [20] in discussing acute fulminating frontal sinusitis, such as that occurring after swimming and characterized by intense frontal pain, generalized headache and spreading edema, advocated frontal trephining through a small incision over the inner angle of the brow. They advised avoiding further surgical intervention until the patient gives evidence of some power to confine or reduce the virulence of the infection and stated that if the patient demonstrates no power of resistance, further surgical efforts will prove futile. Furthermore, they expressed the opinion that in those cases in which a fulminating infection has led to sepsis and meningitis within twenty-four hours after the appearance of symptoms, radical operation favors dissemination of the infection. Lillie [21] also suggested that when it is deemed necessary to intervene surgically in a case of acute suppurative frontal sinusitis, the operation should be in the form of an external incision and a trephine opening in the floor of the sinus. Imperatori [19] agreed that a small external opening should be made in the frontal sinus to drain the infection which follows swimming and diving. He stated that relatively little pus may be present and that the chief finding is apt to be severe congestion of the vessels.

Wilensky [22] remarked that chiseling of the anterior wall of the frontal sinus may produce infection in the diploe of the frontal bone. He expressed the opinion that this complication is less likely to follow an opening made in the floor of the sinus, since in this area the bone is compact and almost never contains diploe. Although he was referring

20. Williams, H. L., and Heilman, F. R.: Spreading Osteomyelitis of the Frontal Bone Secondary to Disease of the Frontal Sinus, Arch. Otolaryng. **25**: 196-207 (Feb.) 1937.

21. Lillie, H. I.: External Operations on the Frontal Sinus, Am. J. Surg. **42**:199-206 (Oct.) 1938.

22. Wilensky, A. O.: Association of Osteomyelitis of the Skull and Nasal Accessory Sinus Disease, Arch. Otolaryng. **15**:805-833 (June) 1932.

to cases of osteomyelitis of the skull, such an extension of infection from the sinus into the diploic veins of the skull may be the forerunner of an intracranial complication. Bucy and Haverfield [12] stated their belief that once the diagnosis of acute fulminating frontal sinusitis has been made the affected sinus should immediately be drained externally. McNally [2] held this view when he reported 4 cases of such sinusitis occurring in young adults during the swimming season. In every instance there was a subperiosteal abscess without a detectable fistula through the bone into the sinus, suggesting extension of infection by venous thrombosis. Because of this finding, he stated that it seemed to be good procedure to remove the posterior wall of the frontal sinus at the same operation and search for an epidural abscess developing by that route. He felt that in external drainage the nasofrontal duct should not be disturbed. In all of McNally's cases surgical drainage was employed and all were complicated by either osteomyelitis of the skull or abscess of the brain. Carmody [23] in discussing McNally's paper stated that in such cases the nasofrontal duct should be opened for better drainage. Faulkner [3] likewise championed conservative treatment by promoting drainage through the natural ostiums. In the event of empyema of the frontal sinus with complete block and severe general reactive symptoms, he advised trephining the frontal sinus and exenterating the ethmoid and sphenoid sinuses completely.

Simpson [1] advised delaying the complete operation until all acute infection had subsided and emphasized that drainage only should be sought in acute fulminating sinus infection. He advised external drainage without an attempt at complete operation in patients in whom orbital and subperiosteal abscesses were developing. Porter [24] advised operation extensive enough to drain adequately all the sinuses involved in these cases and suggested the use of chemotherapy for those patients who have failed to respond to other treatment.

Furstenberg [14] in deprecation of surgical attacks on acutely infected sinuses reported that 42 of his series of 58 patients with osteomyelitis of the skull revealed that there had been a previous operation, either on an acutely infected sinus or on one which was in a stage of acute exacerbation of a chronic infection.

SUMMARY OF OBSERVATIONS IN THIRTEEN CASES

In the past ten years 23 patients with either acute fulminating sinusitis or complications resulting from this disease have been admitted to the University Hospital, and 22 presented evidence of extension of the infection into the calvarium or the intracranial contents. In 13 of

23. Carmody, T. E., in discussion on McNally.[2]

24. Porter, C. T.: Sinusitis: The Present Rationale of Treatment, Ann. Otol., Rhin. & Laryng. **48**:769-774 (Sept.) 1939.

these, the disease was apparently due to swimming. In this group of 10 men and 3 women between the ages of 11 and 33 years severe frontal pain appeared as the first symptom, and there was marked swelling about the eyes in all except 1. In no case was there a history of significant nasal discharge or chronic nasal complaint. Eight patients stated that there was no evidence of a head cold preceding the onset of the disease, while the other 5, so far as the records show, made no statement concerning this. In 2 cases there was suggestive evidence of intracranial involvement within five days after the first symptom appeared; in the others of the series no definite information regarding this point was recorded.

The complications were diffuse osteomyelitis, epidural abscess, subdural abscess and abscess of the frontal lobe of the brain.

Diffuse osteomyelitis developed in 2 cases. One patient was subjected to an intranasal operation four days after the onset of his symptoms and to an external drainage of the frontal sinus two days later. Osteomyelitis developed slowly; operation was performed thirteen weeks after the onset of the symptoms, and fifteen weeks later an abscess in the frontal lobe of the brain was drained. The patient recovered.

The other patient was one in whom severe frontal pain developed, followed by generalized headache within three days. Because of a septic type of temperature curve he was treated with sulfanilamide and then sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) in adequate doses. During the second week, when staphylococci were recovered from the blood stream and it was felt that he had thrombosis of a cavernous sinus, he was given sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole). His temperature returned to normal, and his general condition improved sufficiently so that he could leave the hospital. One month later he returned to the hospital with a recurrence of symptoms and evidence of osteomyelitis of the skull. An operation to excise this extensive osteomyelitic process was performed three months after the onset of symptoms.

Epidural abscess was encountered in 1 case. The patient was a woman. Her symptoms during the first week of an attack of left frontal sinusitis suggested intracranial extension of the infection. In the second week of her illness the frontal pain and nasal discharge ceased, and during the next four weeks she remained free from any evidence of an organic lesion of the central nervous system or signs of suppurative sinus disease. After this, however, she complained of pain and tenderness in the forehead, well above the upper limits of the right frontal sinus. An operation was performed, which revealed a large well encapsulated epidural abscess in the right frontal region.

Subdural abscess developed in 1 case.

Abscess of the frontal lobe of the brain was encountered in 8 cases. In 6 of these the abscess was associated with focal erosion of the

posterior wall of the frontal sinus and an epidural collection of pus in that area. The other 2 cases have been already presented with those in which diffuse osteomyelitis developed over a relatively long period.

The surgical procedures which preceded diagnosis of these complications may be summarized as follows:

Intranasal operations to promote drainage were performed in 3 cases. Since the operations were performed before the patients were referred to University Hospital, the exact technic of these surgical procedures was not recorded in the hospital histories.

In the first case the operation was done three days after the onset of symptoms. This procedure was followed two days later by one-sided convulsions, which occurred one to three times a day during a period of one week. At the end of this time the patient was admitted to the hospital and given sulfanilamide. There was clinical evidence of acute suppurative pansinusitis on the left side. There were no further convulsions after the first day of hospitalization, and his condition improved rapidly until ten days later he was symptom free and showed no evidence of either suppurative sinusitis or organic involvement of the central nervous system. Recovery was spontaneous and complete.

In the second case the operation was done four days after the onset of symptoms. Diffuse osteomyelitis developed slowly, and an operation was performed to relieve this condition thirteen weeks later.

In the third case the operation was done three months after the onset of symptoms. A brain abscess developed subsequently.

External operations on the frontal sinus were performed in 2 cases. In one case external drainage was carried out four days after the onset of symptoms; a subdural abscess ensued. In the other case this operation was performed two weeks after the onset of symptoms, and an abscess subsequently developed in the frontal lobe of the brain.

Drainage of soft tissue abscesses was done in 3 cases. In one instance an operation was performed to drain a subperiosteal abscess over the frontal sinus seven days after the onset of symptoms. Within two weeks there was no clinical evidence of sinus suppuration. A brain abscess became manifest, however, and operation was performed after ten weeks. Three weeks later there was a recrudescence of the infection, which required radical operation on the frontal sinuses. At operation a large epidural abscess was encountered.

In the second case the patient underwent an operation to drain an orbital abscess one week after the onset of symptoms. Nothing further was done in spite of continued evidence of sinus suppuration. Four months later a Killian type of operation was done; at this time an epidural abscess and a small localized area of osteomyelitis above the frontal sinuses were discovered. Sudden death on the second day after operation was due to massive edema in the brain around a small unsuspected abscess of the frontal lobe of the brain.

In the third instance a subperiosteal abscess developed near the inner canthus of the eye three weeks after the onset of symptoms; this was incised and drained as soon as it appeared. Three weeks later a Killian type of operation was done for suppurative sinusitis; at this time there was no evidence of cranial or intracranial extension of the infection.

In 5 cases there was no surgical intervention during the early stages of infection. In 1 case a Killian operation was done six weeks after the onset of symptoms, and a brain abscess was drained four weeks later. In another instance a Killian operation and drainage of an abscess in the frontal lobe of the brain were done simultaneously, six weeks after the onset of symptoms. In another case a Killian operation was done eight weeks after the onset of symptoms, at which time a large epidural abscess was found. In the fourth instance the patient, one of those in whom diffuse osteomyelitis developed, received no treatment except chemotherapy for the continuing sinus suppuration until, at the end of three months, an operation was done for extensive diffuse osteomyelitis of the skull and abscess of the frontal lobe of the brain. In the fifth case no operation was performed. Although there was roentgenologic evidence of osteomyelitis of the anterior wall of the left frontal sinus, spontaneous recovery took place. The patient was seen three months ago, and although he was requested to return again for further examination, he has not presented himself at the clinic.

Bacteriologic studies in these 13 cases demonstrated a growth of staphylococci in all instances except 2; in these there was no bacteriologic report.

All patients recovered except the one mentioned, whose sudden death on the second day after a Killian operation was attributed to massive cerebral edema associated with an unsuspected abscess of the frontal lobe.

COMMENT

In this series of cases it is interesting to note the various types of treatment pursued and the complications which developed. In the group are persons who were operated on early, some who were treated conservatively while being watched intently for the first evidence of extension of infection to the calvarium or cranial contents (which would prompt immediate surgical intervention) and some who seemed to have been neglected for a relatively long time.

One is impressed with the relative rarity of this type of disease. If this condition were a common complication of swimming, there would be many such patients in the hospital during the summer months.

Is the otolaryngologist to operate early by one approach or another on every patient with acute fulminating frontal sinusitis characterized by severe pain, external swelling and fever? If he is to do this, he

may operate on some, and perhaps many, that do not have incipient osteomyelitis or brain abscess. Without invidious intent, I wish to state that at this time there seems to be no definite proof that prompt external drainage has prevented intracranial complications in cases of acute fulminating sinusitis. There are, however, instances in which complete recovery has occurred following incision and drainage of soft tissue abscesses and thorough surgical exenteration of the infected sinuses after subsidence of the acute process. Although a surgical attack on an acutely infected sinus is not the only cause of osteomyelitis of the skull, the association has been recognized for some time, and my colleagues and I are convinced that it is no mere coincidence that 42 of a series of 58 patients with osteomyelitis of the skull were subjected to surgical operation on the sinuses during an acute phase of the infection.

Until the specific manner of development of acute fulminating sinusitis such as that which follows swimming, and the exact pathologic processes which promote rapid dissemination of infection in this disease become known, it is not possible to speak dogmatically of specific preventive therapy.

Several theories have been proposed regarding the manner in which sinus infections develop after swimming; I will present another, based on clinical observation. It is recognized that normal healthy sinuses are essentially sterile and that they are lined by a thin delicate mucous membrane, which under the conditions of health has not been prepared in any way to receive infection such as that which may quickly follow the forcing of virulent organisms from the nose into these cavities during swimming and diving. As in any unprotected tissue, a virulent organism may enjoy rapid growth and produce extensive damage. In the common head cold it is safe to assume that the inflammatory thickening of the nasal mucous membrane extends through the sinus ostiums into the sinus cavities and that this edema and the accompanying cellular infiltration represent a protective response which undoubtedly accounts for the rarity of fulminating sinusitis complicating the common head cold. Apropos of this theory it was interesting to note that in the group presented, no patient admitted having chronic sinusitis or a significant nasal discharge. Similarly, it has not been our experience in the clinic of the University Hospital or in private practice to encounter severe suppurative sinusitis after swimming in patients having chronic hyperplastic sinusitis. This apparent lack of a protective barrier is further substantiated by the occasional rapid extension of infection to the cranial contents. In 2 persons, for example, there were indications of such an extension occurring within five days after the onset of symptoms. The logical explanation for this occurrence is that rapidly progressing septic thrombophlebitis of the perforating veins of the sinus takes place before enough time has elapsed for empyema to develop.

Can one then expect to prevent intracranial complications in such cases by surgical drainage of the frontal sinus? In these particular instances I feel that one cannot.

In the series there were but 2 cases of diffuse osteomyelitis of the skull, and in these the infectious process in the diploe developed slowly over a period of three months. The remainder of the complications were intracranial and associated in most instances with focal erosion of the posterior wall of the frontal sinus. Of course, from this small group of cases it is impossible to draw significant conclusions, but the observations are highly suggestive that the complications of this disease are more likely to appear in the form of epidural abscess, subdural abscess or brain abscess than they are to take the form of suppurative processes involving the diploic structure of the calvarium.

Although I am willing to admit that it might seem sound and necessary, because of intense pain and swelling, to drain an acutely infected frontal sinus, I feel that there is little to be gained by it during the first few days. In this period the infection is either spreading to the cranial contents or localizing in the sinus cavity. If the suppurative process remains confined to the sinus and forms an empyema which does not drain adequately through the nose, a complete operation can be done safely in four to six weeks. If signs of intracranial involvement become evident, a complete operation on the frontal, ethmoid and sphenoid sinuses should be performed, including removal of the posterior wall of the frontal sinus in order to examine the dura over a rather wide area. Thus an early operation has as its objective the relief of the intracranial complications and not primarily the cure of sinusitis. Unless there is a definite goal to be gained by early surgical drainage of an acutely infected sinus, one should not risk the chance of precipitating diffuse osteomyelitis by an inordinate surgical procedure. Of course, it is understood that if there is clinical evidence of spreading osteomyelitis, manifested by an extending pitting puffy edema beyond the borders of the frontal sinuses, removal of all infected bone must be performed, exposing a generous margin of healthy dura on all sides of the osteomyelitic process. My experiences and those of my colleagues in cases of osteomyelitis of the skull have made us believe that this disease is one which in most instances develops rather slowly and shows some tendency to localize. We feel that it is likely that spontaneous osteomyelitis subsequent to acute fulminating sinusitis is in some cases secondary to an epidural abscess.

At the onset of an acute fulminating process in the paranasal sinuses, chemotherapeutic agents may offer valuable aid. McMahon [25] discovered

25. McMahon, B. J.: Influence of Sulfanilamide on Infected Sinuses of Rabbits: Chemical and Microscopic Studies, Arch. Otolaryng. **28**:222-233 (Aug.) 1938.

from chemical and microscopic studies of infected sinuses of rabbits that sulfanilamide could be recovered in considerable amounts from the sinuses of both controlled and infected animals following administration of the drug by mouth, from which he concluded that "sulfanilamide should be as effective in the treatment of streptococcic sinusitis as it is in the treatment of streptococcic infections eleswhere in the body." Since in many instances fulminating sinusitis is of staphylococcic origin, it would seem that sulfathiazole is the therapeutic agent to be used in the early treatment of this condition.

A case in point is that of W. McB., aged 9, recently admitted to the University Hospital. In the absence of head cold or other probable cause, intense pain over the root of the nose, left cheek and left side of the forehead developed twenty-four hours before admission to the hospital. These symptoms were associated with prostration and a temperature of 103 F. Within twenty-four hours, the left eye was swollen shut, and there was intense edema of the nasal mucous membrane on the left side associated with a thin serous exudate in the left middle meatus. Staphylococcus aureus was the invading organism, and sulfathiazole in adequate amounts was administered for three days. The swelling edema and most of the tenderness over the cheek, root of the nose and forehead disappeared in forty-eight hours. Thick purulent exudate which appeared in the left side of his nose during the second day was no longer in evidence at the end of a week.

SUMMARY

A review of recent literature on acute fulminating sinusitis reveals various explanations of the development of sinusitis after swimming. The divergent opinions regarding treatment of this condition are recorded.

Data from the record of 13 patients who in the past ten years were admitted to the University Hospital with acute fulminating sinusitis subsequent to swimming are cited and briefly analyzed.

It is proposed that the cause of acute fulminating sinusitis after swimming is the introduction of virulent organisms into a normal sinus which is completely unprotected and has not had the advantage of preforming any local barriers to the infectious process.

The recorded instances of rapidly appearing signs of intracranial complications in acute fulminating sinusitis make it seem likely that in these cases the intracranial infection is established by the time frank suppuration in the sinus cavity appears. On this basis the rationale of attempting to drain an acutely infected frontal sinus within the first two or three days to prevent intracranial complications is questioned.

It is suggested from the series of cases cited that the early complications of this disease are more apt to be intracranial than calvarial.

Because of the staphylococcic origin of most of these fulminating infections, the use of sulfathiazole at the onset of the symptoms is urged.

SULFANILAMIDE AND ITS DERIVATIVES

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Paul Ehrlich once said that specific chemotherapy was the goal toward which research workers should strive and conjectured that if sufficient effort were expended along this line therapeutic agents for the treatment of all diseases of infectious origin would ultimately be discovered. To add weight to this prophecy, he gave the world arsphenamine (his salvarsan), the basis for all present day antisyphilitic therapy.

After that time little progress was made in chemotherapy until the advent of paraaminobenzenesulfonamide (now known as sulfanilamide) and kindred drugs, which has given new impetus to both experimental and clinical chemotherapy.

HISTORY

Sulfanilamide, contrary to the belief of many, did not appear suddenly as the fruition of one experiment; it was rather the product of numerous investigations extending over thirty years.

Gelmo, in 1908, working with azo dyes, first mentioned paraaminobenzenesulfonamide, but as no experimental or clinical applications resulted from his work, his discovery is of small acclaim. Hoerlein, Dressel and Kothe, in 1909, used the product in the preparation of azo dyestuffs, and in 1911 Sisley and Porcher proved that diaminoazobenzene when introduced into the intestines is split at the double bond by bacterial action.

In 1913 Eisenberg discovered that certain azo dyes had bactericidal powers in vitro, and in 1914 Tchichibabin and Zeide synthesized pyridium (benzeneazo-α-α-diaminopyridine hydrochloride). Scarlet red, or toluylazotoluylazo-β-naphthol, was accepted by the Council on Pharmacy and Chemistry of the American Medical Association in 1915. In 1917 Jacobs and Heidelberger produced metaaminobenzenesulfonamide. In 1926 Ostromyslensky introduced pyridium for the treatment of urinary tract infections and in 1930 produced serenium (ethoxydiaminoazobenzene hydrochloride), which was used in a similar fashion.

Read before the Eastern New York Eye, Ear, Nose and Throat Society, Troy, N. Y., Feb. 6, 1941.

The original prontosil (a hydrochloride of 4-sulfamido-2′,4′-diamino-azobenzene) was synthesized for the dye industry in 1932 by Mietzch and Klarer. Collaborating with them, Domagk in the same year noted the potency of certain dyes in combating streptococcic septicemia in mice. By adding an azonaphthalene ring and including the sulfonamide group a new drug was synthesized—disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate. This was given the name "streptozon" and patented. It was later known by such names as "prontosil soluble," "prontosil," "prontosil 2," "prontosil red," "prontosil S," "prontosil solution" and "D 5214." At present it is sold under the trade name of neoprontosil and has been given the nonproprietary name azosulfamide by the Council on Pharmacy and Chemistry of the American Medical Association.

Domagk, the director of the pathologic laboratory of Elberfeld, Germany, therefore, deserves the credit for having been the first to use the azo compounds containing the sulfonamide radical in the para position in animal experiments, and it was he who first suggested the clinical application of the compounds. For this work he was awarded the Nobel Prize in Medicine for 1939. The experiments of Domagk concerning the bactericidal activity of the original prontosil were then confirmed by the work of Levaditi and Vaismann at the Pasteur Institute in Paris.

The Tréfouëls, Nitti and Bovet, in the fall of 1935, under the guidance of Professor Fourneau, advanced the hypothesis, already suggested in 1919 by Jacobs and Heidelberger, that sulfanilamide is the therapeutically active principle of the "prontosil" molecule and is formed from the prontosils (the original prontosil and azosulfamide) by reduction in vivo. Working on this assumption, they synthesized this product, a white crystalline powder 0.8 per cent soluble in cold water, and called it "streptocide." To this product such names as "sulfamidyl," "prontosil album" and "prontylin" have also been applied.

In 1937 Long and Bliss, while working with azosulfamide at Johns Hopkins University, learned that it could be reduced with the amino acid cysteine, and by carefully controlled experiments they showed that the sulfanilamide resulting from such reduction was bacteriostatic, while azosulfamide was not.

In May 1938 Whitby reported that sulfapyridine (2-[paraamino-benzenesulfonamido]-pyridine), synthesized by Ewins and Phillips, was effective in experimental pneumococcic infections. Shortly thereafter Evans and Gaisford demonstrated in a well controlled series that this drug was also of great value in the control of pneumonia in man. This work was quickly confirmed in this country by Flippin and his associates and somewhat later by other workers. In addition, sulfapyridine was found to be of great value in the treatment of other types of infection and to surpass sulfanilamide in potency in many of them.

The most recently synthesized sulfanilamide derivative is sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole), which has been found to be of particular value in the treatment of staphylococcic infections. Like sulfanilamide and sulfapyridine, it is of aid in combating other infections and with the passage of time and the accumulation of sufficient clinical data may become specific for infections now allocated to those drugs.

At present, then, sulfanilamide, sulfapyridine and sulfathiazole are on the market in this country, and, thanks to the wise policy of American pharmaceutic houses and to the passage of the Food and Drugs Act, are backed by sufficient experimental data to insure their proper employment as chemotherapeutic agents.

MODE OF ACTION

The mode of action of sulfanilamide and its derivatives has not yet been fully elucidated but considerable light has been shed on the problem. In Domagk's original work with the original prontosil its selective action on streptococcic infection was demonstrated in mice. While this drug was efficacious in combating infections of this nature in vivo, it was found to be ineffectual in vitro. Colebrook and Kenny explained this apparent paradox by showing that in vivo the azo dye is broken down into various components, one of which is sulfanilamide, which they considered to be the active principle. Gley and Girard, as well as others, denied, however, that sulfanilamide alone is the active agent and cited as evidence the fact that of two azo derivatives which yield an equal amount of sulfanilamide, one could be shown to exert twice the antibacterial effect of the other.

Brown and associates added supporting evidence by pointing out that excellent results have been obtained with from 40 to 100 cc. of a 2.5 per cent solution of azosulfamide daily, which on analysis yields only 11 Gm. of sulfanilamide per hundred cubic centimeters.

At any rate, whether additional active principles result from the breakdown of azosulfamide in vivo or whether sulfanilamide alone is the active principle, the striking results obtained with these compounds are due to their bacteriostatic action and not to any bactericidal power, and for efficacious therapy, the leukocytes of the body as well as other body defenses are essential. In other words, the chemotherapeutic agent serves merely to retard bacterial growth until the granulocytes, which serve as shock troops, can be mobilized; the final mopping up, as it were, is left to the monocytes and the humoral defenses.

Why sulfanilamide should be more specific for one type of organism, sulfapyridine for another and sulfathiazole for yet another is simply another ramification of this complex problem that needs clarifying.

ABSORPTION AND EXCRETION

Owing to the greater amount of work done on sulfanilamide, considerably more facts concerning its fate in the human body are at one's disposal. On being ingested, it is readily and almost completely absorbed by the gastrointestinal tract, whence it pursues its course via the blood stream throughout the body. Owing to its diffusibility, it easily permeates all the body tissues and is found in most of them in practically equal concentrations. While in the body, a considerable amount of the drug is acetylated, presumably by the liver, and acetylsulfanilamide is eliminated along with the free form by the kidneys. Elimination by normal kidneys takes place fairly rapidly, and, as a consequence, if an adequate concentration in the blood is to be maintained, a four hour dosage schedule must be instituted.

Sulfapyridine, unlike sulfanilamide, is absorbed inconstantly by different patients and by the same patient at different times. Like sulfanilamide, it appears to be fairly equally distributed throughout the body tissues; unlike that drug, it is acetylated more rapidly and to a greater degree, being excreted by the kidneys largely in the acetylated form. Owing to poor solubility, sulfapyridine readily crystallizes out and tends to form renal calculi. This is a real danger and must be rigidly guarded against. Since sulfapyridine is excreted more slowly than sulfanilamide, a six hour dosage schedule will maintain the proper blood level.

Sulfathiazole is rapidly absorbed and rapidly excreted; it is eliminated by the kidneys considerably faster than either sulfanilamide or sulfapyridine. The rapidity of excretion renders it difficult to maintain the proper blood level without relatively frequent dosage. In addition, fluids must be limited to prevent acceleration of excretion to a critical degree. It is generally felt that fluids should be restricted to the point that the daily urinary output fluctuates between 1,000 and 1,500 cc. Unlike both sulfapyridine and sulfanilamide, sulfathiazole does not readily enter the meninges and consequently is of less value therapeutically in treating the various meningitides.

TREATMENT

As was to be expected, most of the clinical reports that appeared immediately after Domagk's initial work on the treatment of streptococcic infections in mice with the original prontosil dealt with infections in which Streptococcus was the invading organism. It was not long, however, until the drug was found to be of value in treating other conditions, and since that time a voluminous literature on the subject has accumulated. Sufficient data are now at hand to allow evaluation of the potentialities of sulfanilamide and its derivatives and selection therefrom of the drug most apt to be efficacious in a specific infection. For this

reason it is of paramount importance to learn the causal agent as soon as possible, though therapy may be instituted at once, should clinical evidence be sufficiently strong.

Sulfanilamide is still the drug of choice when the virulent organism is a hemolytic streptococcus.

Sulfapyridine is most effective in controlling pneumococcic infections (such as mastoiditis, meningitis, otitis media, peritonitis, pneumonia and acute sinusitis), staphylococcic meningitis and all gonococcic infections.

Sulfathiazole is definitely preferred when the invading organism is Staphylococcus, except in cases of staphylococcic meningitis, in which sulfapyridine is preferred. The reason for this may be found in the aforementioned fact that sulfathiazole reaches the meninges in concentrations that are therapeutically ineffectual. Sulfathiazole is as valuable as sulfapyridine in the treatment of pneumococcic pneumonia.

In each instance, however, there are insufficient data at hand to assure one that the specific drug will be therapeutically effective. With repeated clinical trials in the future, the treatment of such infections may be placed on a more dependable basis.

Whenever sulfanilamide or one of its derivatives is employed, it is essential that the patient receive in addition the supportive therapy indicated as well as any surgical intervention that may become necessary. If abscesses form, drainage should at once be instituted, as these drugs will in no wise alter the course of the disease so long as collections of pus remain. This is of particular importance if the invading organism is Staphylococcus, whose propensity for causing abscess formation is well known. It is of particular importance to the otolaryngologist to remember that where there are destruction and necrosis, these drugs are ineffectual, and the diseased tissue must be removed if cure is to be effected. Another point which needs repeated emphasis is the fact that these drugs are insidious in their ability to mask symptoms of bone involvement until dire complications have set in. In fact, roentgenographic evidence may be completely lacking in cases of severe mastoiditis, which, when administration of the drug is stopped, assumes its classic form both clinically and roentgenologically.

For certain types of infection, in addition to treatment with sulfanilamide or one of its derivatives, specific therapy along biologic lines is important. Type-specific antiserum, either horse or rabbit, should be used in cases of severe lobar pneumonia or, for that matter, any type of severe pneumococcic infection. When the invading organism is Clostridium welchii, the use of antitoxin in addition to sulfanilamide is of great value and may be the deciding factor in a given case. Should meningitis caused by Haemophilus influenzae (particularly of the beta type) appear, sulfapyridine is to be used, but the mortality rate is halved

by auxiliary use of antiserum. In scarlet fever the concomitant use of antitoxin is said to lower the incidence of sequelae considerably.

These drugs also have prophylactic uses. Frequently otitis media can be aborted at an early stage by prompt and adequate sulfanilamide therapy, but since this drug frequently masks signs and symptoms, we do not use it until the drum has been opened.

According to some, the routine use of sulfanilamide in the treatment of patients who have undergone operations for appendicitis has considerably lowered the mortality rate.

There is at present little in the literature concerning the local application of sulfanilamide and its derivatives. Jensen and associates have reported excellent results in the treatment of compound fractures. In a series of 39 cases there were only two infections, and these resulted when the fractures were compounded. Lane and Vinson have successfully treated aphthous stomatitis by local application of sulfanilamide, and Long has had considerable success in treating burns, streptococcic ulcers and indolent lesions caused by Staphylococcus. The latter advises the use of a powder atomizer in applying the agent to the affected area.

TOXIC EFFECTS AND UNTOWARD REACTIONS

Sulfanilamide and its derivatives, in spite of their great therapeutic effectiveness in many conditions, have not been without toxic manifestations. These vary in frequency and seriousness, depending on many factors, such as the individual patient, the type of drug employed, the concentration of the drug and the type of infection.

Cyanosis is frequently observed with sulfanilamide and sulfapyridine and infrequently with sulfathiazole. It is due in most cases to an accumulation of methemoglobin, although traces of sulfhemoglobin are generally present also. It should occasion no alarm, nor should the treatment be stopped because of its presence. The intravenous use of thiamine hydrochloride, which has a dye base similar to the leuko base of methylthionine chloride (methylene blue), will return the blood pigment to normal within ten minutes, but unless cyanosis becomes extreme, this measure is not indicated.

The myth that all drugs containing sulfur or sulfur radicals and such dietary items as eggs are contraindicated during sulfanilamide therapy should be exploded once and for all. There is no evidence for the idea that these increase the formation of sulfhemoglobin, but any type of laxative, whether it contains sulfur or not, will increase the amount of circulating sulfhemoglobin. Hence purgatives should be used only when absolutely necessary.

Nausea and vomiting are frequently seen with sulfapyridine, occasionally with sulfanilamide and rarely with sulfathiazole. They may

often be relieved by giving the drug well disguised in milk or some other liquid medium, but if the attacks are severe, this is of no avail. The use of phenobarbital in doses of ½ or ¼ grain (0.03 or 0.015 Gm.) one-half hour before the administration of the drug often reduces nausea and vomiting. Should these symptoms become too severe, the administration of the drug may have to be discontinued or another drug employed.

Dizziness is frequently noted both with sulfanilamide and sulfapyridine but rarely with sulfathiazole. It is of no import.

Psychoses occurring early and rather frequently are encountered when sulfapyridine or sulfanilamide is used, but infrequently when sulfathiazole is employed. When these occur, unless the gravity of the infection dictates the continued use of the drug, its use should be stopped, and fluids forced.

Peripheral neuritis has rarely been seen with sulfanilamide, never with sulfapyridine and infrequently with sulfathiazole. Several cases were reported when sulfamethylthiazole (2-[paraaminobenzenesulfonamido]-4-methylthiazole) was employed, but this drug has now been withdrawn from the market. Whenever this symptom occurs, it is imperative to stop giving the drug and force fluids.

Acidosis has occasionally been noted when sulfanilamide was used but not when other drugs were given. Should it occur, it is easily treated by the administration of adequate amounts of sodium bicarbonate. Contrary to earlier teachings, it is not necessary to give sodium bicarbonate with each dose of sulfanilamide, as acidosis is a rare complication.

Fever may be seen with all the drugs, usually between the fifth and ninth day, but occasionally later. It is seen in 10 per cent of the patients receiving sulfanilamide, in 4 per cent of those receiving sulfapyridine and in 10 per cent of those receiving sulfathiazole. In such instances, cessation of treatment with the drug and forcing of fluids are advisable.

Rash, which may be of any variety, is generally seen from the fifth to the ninth day in 1.9 per cent of patients treated with sulfanilamide, in 2 per cent of those treated with sulfapyridine and in 5 per cent of those treated with sulfathiazole. The drug should be withdrawn. Since patients taking sulfanilamide not infrequently become sensitive to sunlight, they should avoid ultraviolet rays as this is one of the commonest causes of rash.

Hepatitis is a severe toxic manifestation, and death not infrequently results. It is seen in about 0.6 per cent of patients receiving sulfanilamide, rarely in those receiving sulfathiazole and, according to reports, never yet in those receiving sulfapyridine. There is an icteric tint to the skin and scleras, with an increase in the icterus index. Impending hemolytic anemia, which occurs not infrequently with both sulfanilamide and sulfapyridine but rarely with sulfathiazole, may be heralded by

icteric scleras and pale mucous membranes. In both instances, the drug must be stopped at once and fluids forced. Should the anemia be severe, a blood transfusion is indicated.

Leukopenia with granulocytopenia occurs infrequently with all the drugs, but because of its serious nature must be obviated. Sore throat may be the first complaint, and whenever it is present in a person receiving these drugs, it should not be lightly dismissed. Should this state supervene, administration of the drug must be halted, fluids forced, and supportive therapy instituted. Pentnucleotide has been found of great value in treating conditions of this character. Frequent blood counts are essential for all patients receiving sulfanilamide or one of its derivatives; this cannot be emphasized too much.

Hematuria has not been reported in patients treated with sulfanilamide but is seen frequently in those treated with sulfapyridine and not infrequently in those treated with sulfathiazole. In most instances it is due to precipitation of the acetylated form of the drug in the renal tubules. Should the use of the drug be continued, large calculi may form, giving rise to anuria with azotemia, an extremely serious condition. Whenever hematuria occurs, the drug must be withdrawn and fluids forced. However, should the condition progress to anuria with azotemia, a urologist must be consulted, and ureteral catheterization resorted to. To prevent precipitation of the acetylated drug in the first place, sufficient fluid to insure a urinary output of from 1,000 to 1,500 cc. per day must be given. Some persons appear to conjugate these drugs more rapidly than others, and what appears to be adequate fluid intake for most will be insufficient for them.

Injection of scleras and conjunctivas may occur between the fifth and the ninth day along with rash in patients receiving sulfathiazole. This has not yet been reported with either of the other two drugs. When the condition occurs, the drug should be stopped and fluids forced.

Purpura haemorrhagica has been reported as occurring rarely in patients receiving sulfanilamide and sulfapyridine and never in those receiving sulfathiazole. Ocular and auditory disturbances occur rarely with all three, and painful joints have been reported with sulfathiazole. Should any of these untoward reactions be observed, cessation of administration of the drug and forcing of fluids are indicated.

Gastrointestinal disturbances other than nausea and vomiting are rarely seen and are usually not severe.

It should be added that the development of any untoward reaction as a result of taking sulfanilamide or one of its derivatives is an individual phenomenon, and whether the drug is to be continued or not must be decided by the attending physician after evaluating the many factors at hand.

DOSAGE

In a recent article Long has admirably summarized the dosage schedule to be followed with sulfanilamide and its derivatives. The schedule he recommends is as follows:

When sulfanilamide is to be employed in the treatment of severe infections, the initial dose (oral) should be 0.10 Gm. per kilogram of body weight. Subsequent doses (oral) should equal a total daily dose of 0.10 Gm. per kilogram, which should be divided into six parts and given every four hours, night and day, until seven days of normal temperature have elapsed. For mild and moderately severe infections there should be a total daily dose of 0.10 Gm. per kilogram of body weight, divided into six doses and given every four hours, night and day, until five days of normal temperature has elapsed. Blood determinations should be made, particularly in severe infections, and a concentration of from 8 to 10 mg. per hundred cubic centimeters of the free form should be maintained.

When sulfapyridine is to be used, the initial dose should be 4.0 Gm. Subsequent doses should be 1.0 Gm., given every six hours, night and day, until the temperature has been normal for seventy-two hours. For infants and children the initial dose is 0.15 Gm. per kilogram up to 25 Kg. of body weight. The subsequent daily dose should be 0.15 Gm. per kilogram, divided into four parts and given every six hours, night and day, until the temperature has been normal for forty-eight hours. It is of great importance to observe the blood sulfapyridine level of the total and of the free form. Since the free form is the only therapeutically active portion, it should be kept between 8 and 10 mg. per hundred cubic centimeters. However, owing to the propensity of this drug to form calculi, the total should never exceed from 10 to 14 mg. per hundred cubic centimeters. It is in those cases in which conjugation proceeds rapidly that formation of calculi will occur, and these cases can be diagnosed only if care is taken to determine both the free and the total blood concentration.

If sulfathiazole is to be used, the dosage in most cases is similar to that for sulfapyridine. However, in severe staphylococcic infections, such as cellulitis and lymphangitis, after an initial dose of 4 Gm., 1.5 Gm. should be given every four hours, night and day, for seven additional days. For staphylococcic bacteremia in adults, the initial dose should be 4 Gm. followed by 1.5 Gm. every four hours, night and day, until the temperature has been normal for forty-eight hours. For the next fourteen days a constant dose of 1 Gm. every four hours, night and day, should be employed, and 0.5 Gm. every four hours for another two

weeks. For infants and children the dose should be determined according to Young's rule:

$$\frac{\text{Age of child in years}}{\text{Age in years} + 12} = \text{Child dose}$$

The blood concentration should be observed as with sulfapyridine and sulfanilamide.

The oral route should be used with all these drugs when possible.

Up to this point, we have been dealing with the general indications and uses of sulfanilamide and its derivatives. Before citing the conclusions, we should like to bring to attention a series of 16 cases of true meningitis, the result of otitic complications.

This series one of us (D. S. C.) reported at the Ninetieth Annual Session of the American Medical Association in St. Louis in May 1939,[1] and we believe it still is one of the largest reported up to the present time. In general the treatment consisted of radical surgical intervention (namely, complete mastoidectomy with exposure of the middle and posterior fossae and crucial incision of the dura over this area to encourage free drainage of spinal fluid) and the intensive use of sulfanilamide, the only compound of the group known at that time. Of the patients in this series, all with positive cultures on two or more examinations of the spinal fluid, 12 recovered and 4 died, a total of 75 per cent recoveries; before the use of these drugs the normal expectancy of recoveries was 2 to 4 per cent.

These 16 patients were seen in all stages of the disease, and the majority of them showed full blown and apparently hopeless meningitis at the first examination. We are convinced that this large percentage of recoveries was the result of radical but indicated surgical procedure and the use of sulfanilamide and feel that one without the other could never have effected so many recoveries.

In the cases in which the patients recovered, the bacteriologic findings were: Streptococcus haemolyticus, in 7; Pneumococcus type III, in 1; Pneumococcus type IX, in 1; Pneumococcus type XXIX, in 1; Staphylococcus aureus, in 1; Bacillus influenzae, in 1. In the 4 cases in which the patients died, the bacteriologic examinations revealed Pneumococcus type I in 2, Pneumococcus type III in 1 and Str. haemolyticus in 1.

It is possible that if we had had present knowledge and, in addition, sulfapyridine and sulfathiazole, these results might have been even better.

SUMMARY AND CONCLUSIONS

The history, mode of action, absorption and excretion, therapeutics, toxic manifestations and dosage of sulfanilamide and its derivatives are reviewed.

1. Cunning, D. S.: Treatment of Otitic Meningitis, Arch. Otolaryng. **30**:950-972 (Dec.) 1939.

These compounds, when properly used, constitute a valuable therapeutic agent in the treatment of many infectious diseases.

There are four rules to be followed in administering these drugs: (1) isolate the organism; (2) make sure the diagnosis is correct; (3) give large doses boldly and consistently; (4) be on the lookout for bad effects.

The only real contraindication for the drug is previously known sensitivity. Sensitivities seem to be on the increase.

These drugs are bacteriostatic and not bactericidal.

Abscesses or pus should always be drained.

Sulfanilamide and sulfathiazole should be given every four hours; sulfapyridine, every six hours.

The drug should be kept at the proper blood level (8 to 10 mg. per hundred cubic centimeters). A complete blood count should be taken every forty-eight or seventy-two hours. The urine should be watched for blood and crystals.

The patient should be kept in bed; if he is sick enough to require the drug, he is sick enough to be in bed. Start administration of the compound as early as possible. If the patient has nausea and vomiting, phenobarbital in doses of ¼ or ½ grain (0.03 or 0.015 Gm.), given one-half hour before the drug, helps in most cases.

Sulfanilamide seems to work best in cases in which the invading organism is Streptococcus haemolyticus. Sulfapyridine is best for pneumococcic and sulfathiazole for staphylococcic infections. Sulfathiazole seems to be the best all around drug at present, as it acts on Pneumococcus, Streptococcus, the gonococcus, Bacillus coli and Staphylococcus.

Staphylococcus is the hardest of all to treat with chemotherapy, owing to its tendency to form a large amount of pus.

These drugs work well as adjuncts to medical and surgical treatment of acute infections, but they are of little value in the treatment of chronic infections.

Sulfathiazole helps greatly in the treatment of cellulitis and erysipelas of the nose and the face. It is practically useless in any form of meningitis, as less than 5 per cent of it permeates the spinal fluid. The other two drugs, however, enter the spinal fluid freely.

If the desired effect is not produced by these drugs, it is helpful to ask the following questions: Is the diagnosis correct? Are any complications present? Is the patient getting a sufficient quantity of the drug? Is there an overwhelming infection with, for example, accumulation of pus or necrotic bone in which surgical intervention will help?

121 East Sixtieth Street.

Case Reports

FOREIGN BODIES WHICH I HAVE NOT REMOVED

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The interest in endoscopic procedures is today greater than ever before. The results, particularly in the removal of foreign bodies from the food passages and airways, have been so spectacular that there is scarcely a well organized nose and throat clinic that has not accumulated a collection of weird and common objects of assorted sizes and shapes, which have been removed from these passages.

Recently there appeared a review of nearly 1,000 cases in which foreign bodies had been removed, with a mortality of only 1.68 per cent. This is indeed a tribute to the science of endoscopy and to the knowledge and skill of the individual operators.

In endoscopy, as in all successful therapeutics, there is always the danger of overenthusiasm. Thinking that a counterweight may not be amiss, I am reporting these somewhat unusual cases.

REPORT OF CASES

CASE 1.—A white man accidentally swallowed some loosened upper dental bridgework. On two successive days, with the patient under anesthesia induced with avertin with amylene hydrate, esophagoscopic examination was performed, but the foreign body was not seen by the operator.

On the third day following the accident I was called to see the patient. At the hospital I was told that he had swallowed a loosened upper denture as he was taking a tablet. The roentgenogram showed a shadow which was unlike that of any denture I had ever seen (fig. 1 *A*) but which could possibly be that of an orthodontic appliance. It seemed to be a little too far to the left to be in the esophagus unless it were in a diverticulum.

I passed the esophagoscope down to the cardia, and, having made sure that there was no foreign body in the esophagus, I withdrew the instrument. As the patient was perfectly conscious, I showed him the roentgenogram and asked if the foreign substance in any way resembled the denture he had lost.

"Is that what you doctors are looking for?" he cried in amazement. "Why, that's the wiring on a collar bone and rib I broke ten years ago."

New roentgenograms were made of his chest and abdomen. The denture and loosened clasp were seen lodged in the lower part of the intestinal tract (fig. 1 *B*). The next day these were passed per rectum.

Comment.—This case illustrates the importance, so frequently emphasized by Chevalier Jackson, of taking roentgenograms to include the entire neck, chest and abdomen. Had this been done in the first place, both the denture and the wired clavicle would have been seen and the operator would have been led to elicit the history more accurately. The case also demonstrates the advantage of doing endoscopic work with topical anesthesia, so that if necessary the patient may be questioned.

CASE 2.—A young Negro boy was admitted to the hospital with a history of having "swallowed a screw driver," from which the wooden handle had been removed.

The mother said that the child had suffered a severe coughing spell and that there had been some wheezing at the time of the accident. Otherwise the child had been perfectly comfortable.

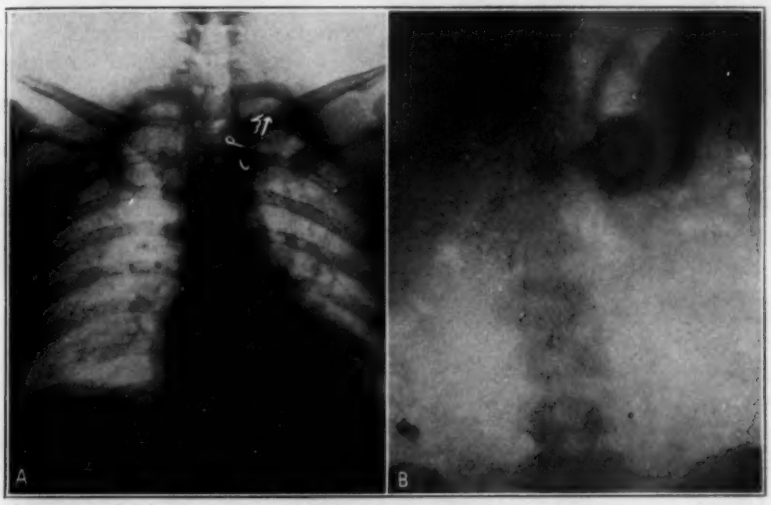

Fig. 1.—*A* shows the old wiring on the clavicle and rib; *B*, the denture on the right side of the lower part of the intestinal tract.

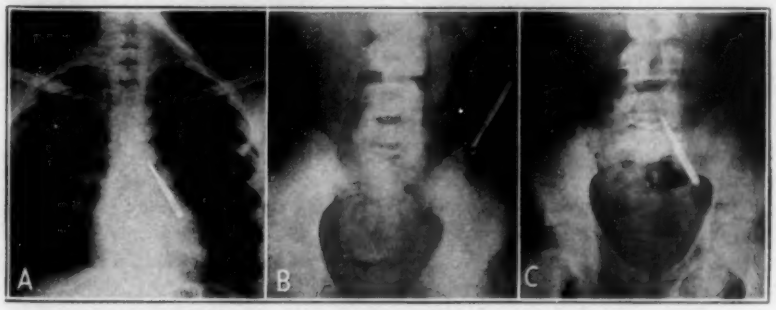

Fig. 2.—*A* shows the screw driver in the left bronchus; *B* and *C*, the screw driver in various parts of the intestinal tract.

Roentgen examination of the chest and abdomen revealed the metal portion of a screw driver in the left main bronchus (fig. 2*A*).

About three hours later a bronchoscopic examination was made. The bronchi on the left side were thoroughly explored, but the screw driver was not seen. In consideration of the possibility that the foreign body had been coughed up and reaspirated into the right lung, the right bronchi were explored. No foreign body was in sight there either.

Further roentgen examination showed the screw driver in the intestinal tract (fig. 2 *B*). Apparently the foreign body had been coughed up and, incredible as it seems, was then swallowed.

The patient was sent back to the ward, and the progress of the foreign body through the intestinal tract was watched by daily roentgenograms (fig. 2 *C*). Three days later the screw driver was passed per rectum, and one week later the patient was permitted to go home.

Comment.—Not infrequently, by the time a patient gets to the operating room a foreign body which has been aspirated or swallowed will have been dislodged from its original site and will be found in some other part of the esophagus or tracheobronchial tree, usually lower down. Occasionally the object will be regurgitated or coughed up during this interval. It is therefore advisable to take a roentgenogram or to examine the patient fluoroscopically immediately before endoscopic manipulation.

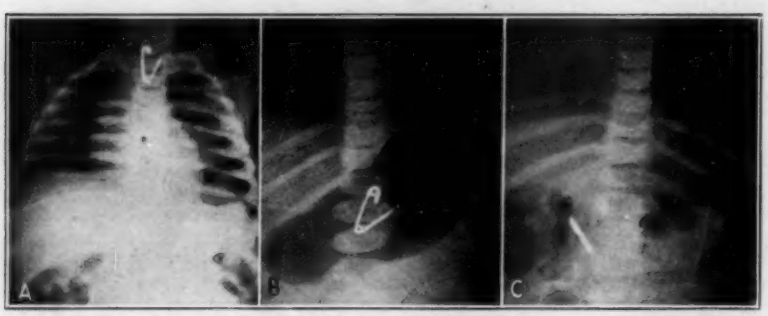

Fig. 3.—*A* shows the open safety pin in the esophagus; *B*, the open safety pin in the stomach to which it had already descended by the time the child was brought into the hospital; *C*, the safety pin in the stomach after its closure through the wall of the stomach.

Case 3.—A child of 8 months was having its diaper changed when the doorbell rang. The mother placed the child and the unadjusted diaper in the crib, stuck the open safety pin into the mattress and went to open the door. When she returned, the safety pin had disappeared. The mother made a thorough search of the room. Not finding the pin and noticing that the child had a slight irritative cough, she began to suspect that the child might have put the pin into its mouth. On palpating the infant's mouth she did not find the pin.

The family physician concurred with the probability that the child had aspirated or swallowed the safety pin. He sent the child for roentgen examination, and the film disclosed an open safety pin with the point up, in the upper part of the esophagus (fig. 3 *A*). The necessity for endoscopic removal of the pin was explained to the parents, who then consented to move the child to the hospital.

When I was called, three hours had elapsed since the accident, and so before esophagoscopy was attempted, another roentgenogram was taken. This showed that the pin had already descended into the stomach (fig. 3 *B*).

The problem of procedure was now for me a difficult one. I was aware of the dangers inherent in allowing such a foreign body to remain in the gastrointestinal

tract, and I was loath to attempt gastroscopic removal. In my service at the Harlem Hospital I have seen a fair number of older children and adults who have accidentally swallowed open safety pins. These patients were carefully watched by frequent roentgenograms, and every one of them passed the foreign body without ensuing complications.

I felt that in so young a child an open safety pin of this size might have difficulty in getting past the pylorus and that conservative surgical intervention would be safe. I called for the assistance of Dr. J. A. Taferner.

A vertical incision was made in the upper portion of the right rectus muscle, just underneath the ribs; the muscle fibers were separated and the peritoneum carefully opened. The stomach was brought into view, and with the aid of two

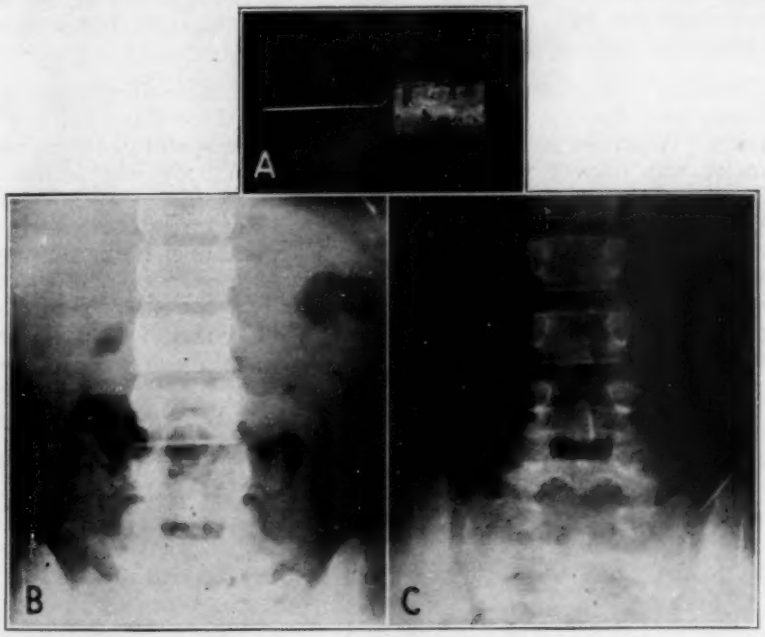

Fig. 4.—*A* shows the dart after it had been passed per rectum; *B*, the dart floating in the stomach, just below the air bubble (not very clearly reproduced); *C*, the dart in the lower part of the intestinal tract.

pieces of gauze, to prevent the stomach from slipping from the grasp, the pin was felt through the intact stomach wall. The keeper was grasped, and by gentle manipulation the pin was closed. The abdominal wound was sewed up in layers.

The child made an uneventful recovery. There was no elevation of pulse or of temperature, and it took its feedings on uninterrupted schedule. Roentgenograms were taken to watch the progress of the closed pin (fig. 3*C*), which was passed per rectum on the third day following the operation.

CASE 4.—A boy of 12 swallowed a dart made of a straight needle inserted into a cork (fig. 4*A*). His family physician took a roentgenogram. This showed the needle and cork in the fundus of the stomach (fig. 4*B*). The boy was

advised to take quantities of fluids and bulky soft foods, such as banana and mashed potato, with the idea of shielding the point of the pin or coating the intestinal tract.

On the third day following the accident I was consulted, because the pin and cork were still lodged in the fundus of the stomach. The child was not ill and he had no definite pain, but he was overwrought and complained of a sense of pressure in the epigastrium.

He was sent home with instructions to the mother to withhold all food and fluids from him until the next day and was told to come back for roentgen examination. A tablet of 1/600 grain (.00012 Gm.) of atropine sulfate was prescribed, to be placed under the tongue three times daily and if necessary washed down with a minimum amount of water.

When the child returned the next day the foreign body had passed into the small intestine (fig. 4*C*). A soft bland diet was ordered. Three days later the dart was passed per rectum.

Comment.—It occurred to me that the cork because of its buoyancy had been kept floating in the fundus as long as there was food in the stomach. When the stomach was emptied and the spasm of the pyloric sphincter was relaxed with atropine, the foreign body was permitted to enter the intestines, where peristalsis did the rest.

Contrary to what might be expected, large blunt foreign bodies which may lodge in folds of the mucosa in the gastrointestinal tract, are more likely to perforate than sharp-pointed objects. Fine-pointed objects, such as needles and pins, are apparently so easily propelled, or feathered, by peristalsis that the point tends to stay in the lumen of the intestines rather than to embed in the wall.

667 Madison Avenue.

INTRACRANIAL EDEMA FOLLOWING OCCLUSION OF ONE LATERAL SINUS

FRANK H. MAYFIELD, M.D., CINCINNATI

Such terms as "serous meningitis," [1a] "otitic hydrocephalus," [1b] "toxic hydrocephalus," "pseudo-abscess" and "cerebral edema of unknown cause" [1c] are among those that have been used to describe a state of intracranial hypertension which occurs in the presence of infection about the middle ear and mastoid process of the temporal bone with no evidence of intracranial infection. The number and variability of these titles indicate that the nature of this state is not well understood. In general the cases which have been reported under these titles can be divided into two groups: those in which there is an increase in the cell count of the cerebrospinal fluid; those in which the fluid shows no rise in the cell count.

The condition in the first group may be explained by an aseptic intracranial inflammatory process secondary to an extradural infection or by mildly active intracranial infections in patients who have sufficient local resistance to prevent suppuration either in the cerebrospinal fluid or in the brain tissue. In the other group, however, in which there is intracranial hypertension with choked disk and headache without cellular reaction in the spinal fluid, this explanation is not adequate.

Ventrilograms of patients with this syndrome usually show ventricles that are normal or smaller than normal, indicating that the increased pressure is due to excessive fluid in the brain itself rather than in the subarachnoid or ventricular spaces.

The case reported here is that of a patient in whom acute intracranial hypertension developed without evidence of intracranial infection following occlusion of the right lateral sinus and right jugular vein. The observations in this case suggested that venous obstruction was responsible for the symptoms of the patient.

Ecker, Ayer and O'Connor [1d] recently reported a case in which venous stasis was the cause of acute intracranial hypertension. They stressed the importance of elevating the head as a help in treatment.

From Department of Surgery, Jewish Hospital.

Read before the Section on Eye, Ear, Nose and Throat of the Ohio State Medical Association, Cincinnati, May 15, 1940.

1. (*a*) Davidoff, L. M., and Dyke, C. G.: Series of Cases of Serous Meningitis, Arch. Neurol. & Psychiat. **36**:1376-1380 (Dec.) 1936. (*b*) Williams, H. L.: Otitic Hydrocephalus, Arch. Otolaryng. **25**:632-652 (June) 1937. (*c*) Sahs, A. L., and Hyndman, O. R.: Intracranial Hypertension of Unknown Cause: Cerebral Edema, Arch. Surg. **38**:428-442 (March) 1939. (*d*) Ecker, A. D.; Ayer, W. D., and O'Connor, F. J.: Increased Intracranial Pressure Attributed to Venous Obstruction, J. A. M. A. **114**:1440-1442 (April 13) 1940. (*e*) Kerrison, P. D.: Diseases of the Ear, ed. 4, Philadelphia, J. B. Lippincott Company, 1930, p. 387. (*f*) Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922, pp. 32-34. (*g*) Kopetzky, S. J.: Otologic Surgery, New York, Paul B. Hoeber, 1925, p. 324.

REPORT OF A CASE

A. S., aged 25, a student nurse, had had numerous admissions to the Jewish Hospital over a period of two years for pharyngitis, tonsillitis and sinusitis. On Sept. 27, 1939 she was again admitted with acute otitis media on the right and was treated by incision of the drum. She was dismissed on October 10 with slight purulent discharge from the right ear. Four days later, on October 14, she was readmitted with pain over the right mastoid process and with clinical and roentgenographic evidence of acute mastoiditis on that side.

The following morning a radical mastoidectomy was done on the right side. There was a large amount of pus in the mastoid tip, and the dural plate was removed, exposing the knee of the lateral sinus. A culture of the pus showed Streptococcus haemolyticus, and sulfanilamide therapy was instituted. The patient continued to complain of pain about the right ear, and the temperature remained slightly elevated. On October 20 she had two hard shaking chills, and there was tenderness over the right jugular vein.

That night the mastoidectomy wound was reopened, and the lateral sinus opened with the expectation of finding a thrombus. There was decided thickening of the wall of the sinus, but when the sinus was opened, there was free bleeding from both ends. Gauze packing was used between the dura and bone and in the sinus to control the hemorrhage. The internal jugular vein in the neck was then exposed and ligated.

After this the patient's fever subsided, but she began to complain of a sensation of fulness in the head. By October 25 she was suffering from considerable headache but showed no focal neurologic signs. Her optic disks at this time showed early papilledema. An attempt to remove the packing from the lateral sinus was made, but copious hemorrhage followed its removal, and the packing was replaced. The patient continued to complain of headache, of a sensation of fulness in her head and of blurred vision. Nevertheless, throughout her illness she was remarkably alert and presented an appearance of well-being. Her temperature remained normal, and at no time was there any opisthotonos.

A lumbar puncture on October 28 released clear fluid under 470 mm. of pressure. Compression of the left jugular vein caused a rise of only 30 mm. of water; compression of the right caused no change.

On October 29 the patient was still alert, perhaps slightly euphoric, but complained of moderate headache. The optic disks then showed 4 D. of swelling bilaterally with hemorrhages into both retinas. There was an equivocal Babinski sign on the left. Otherwise, her neurologic examination showed her normal. Lumbar puncture was repeated on this date. The pressure was 450 mm. of water; the fluid was clear and colorless, and contained no cells. The culture yielded no growth. The protein content was slightly elevated, 50 mg. per hundred cubic centimeters. The effect on the spinal fluid pressure of compression of the jugular vein was not recorded.

After considerable debate as to the advisability of obtaining ventriculograms, it was finally decided in view of the patient's excellent general state to defer operation pending further developments. This seemed especially logical in view of Iglauer's [2] suggestion that occlusion of the sinus might be responsible for the increased intracranial pressure. Then, too, the patient was subjectively better.

On November 2 the gauze packing was removed from the mastoidectomy wound without bleeding. Before this, however, the patient's headache had been less, and within forty-eight hours the spinal fluid pressure was 140 mm. of water,

2. Iglauer, S.: Personal communication to the author.

and the choked disks were subsiding. Unfortunately, the result of the Toby-Ayer test was not recorded on this occasion. Thereafter, her convalescence was uneventful, and she was discharged.

COMMENT

It seems reasonable to assume that the intracranial hypertension was due to venous stasis, for the Toby-Ayer modification of the Queckenstedt test showed that there was only a 30 mm. rise of spinal fluid pressure following compression of the left jugular vein with the right side already occluded. One would expect a far greater rise.

The problem of diagnosis in this case was largely one of excluding the possibility of brain abscess; this was eliminated by the complete absence of cellular reaction in the spinal fluid and the patient's state of well-being. Had she not begun to show improvement when she did, it is likely that ventriculograms would have been taken, and perhaps a subtemporal decompression made.

It has long been established that the right lateral sinus is the predominant channel of venous return from the skull in the majority of persons. Woodhall [3] in 1936 examined 100 consecutive autopsy specimens for anatomic variations of the venous sinuses of the skull with special attention to the torcular Herophili and the two lateral sinuses. The most important observations made in this study were that the right lateral sinus was predominant in 29 per cent of his specimens, that the left side was predominant in 13 per cent and that there was a major disproportion between the two sides in 24 per cent. There was inadequate cross circulation at the torcular Herophili in 10 per cent, and complete absence of one lateral sinus in 4 per cent.

These figures show that in approximately 25 per cent of patients the venous return from the skull is impeded when the predominant side is obstructed either by a thrombus or by surgical ligation. The inadequacy is extreme in from 4 to 10 per cent of patients. From these figures it may be seen that intracranial edema will result in certain instances if the major sinus is occluded and that fatality may ensue if it is occluded in 1 of the 4 per cent of patients who have only one lateral sinus.

In his study Woodhall also noted that the grooves in the skull created by the lateral sinus varied definitely with the size of the sinus. With this in mind Woodhall and Seeds [4] attempted to correlate the variations in size of the lateral sinus as shown on the roentgenogram with the capacity of the sinus itself. It would appear that considerable significance may be attached to this roentgenographic evidence.

At the time they wrote, Woodhall and Seeds [4] were able to find some twenty references in the literature in which the authors suggested that occlusion of one lateral sinus might produce increased intracranial pressure. The most important of these was by Meyer and Ersner,[5] who

3. Woodhall, B.: Variations of the Cranial Venous Sinuses in the Region of the Torcular Herophili, Arch. Surg. **33**:297-314 (Aug.) 1936.

4. Woodhall, B., and Seeds, A. E.: Correlation Between Roentgenograms of the Occipital Bone and the Queckenstedt (Toby-Ayer) Test, Arch. Surg. **37**: 865-870 (Dec.) 1938.

5. Meyer and Ersner, cited by Woodhall and Seeds.[4]

reported 4 cases in which they correlated the dynamics of the cerebrospinal fluid with the operation performed and the skull markings of the sinuses as shown on roentgenograms. One of the patients died, and at autopsy it was demonstrated that there was only one lateral sinus, and that had been occluded. There was no intracranial infection. With few exceptions the patients of this type whose cases have been reported by other authors [1] either had one lateral sinus or jugular vein ligated or showed symptoms suggesting infectious thrombosis of the sinus.

From the study of the case reported here it would appear that one is justified in making a diagnosis of venous stasis in cases of this type when the patient shows intracranial hypertension without cellular reaction in the spinal fluid and a state of general well-being not in keeping with intracranial hypertension due to intracranial infection. This is especially true if it can be demonstrated by the Toby-Ayer modification of the Queckenstedt test or by observations at operation that one lateral sinus or jugular vein has been occluded.

In certain instances the collateral circulation may be adequate to compensate after a period of time; in that event the patient will recover spontaneously. In others decompressive measures may be required to protect the patient's eyes from atrophy as a result of choking of the disks and to save life. Death might follow occlusion of the lateral sinus in the rare instances in which the opposite lateral sinus is entirely absent. It is therefore suggested that before ligation of a jugular vein or a lateral sinus is carried out the presence of a patent and adequate venous channel on the opposite side should be demonstrated. It is also advised that, whenever possible, the clot should be removed from the lateral sinus in cases of infectious thrombosis in preference to ligating the jugular vein.

DERMOID CYST OF THE NASAL BRIDGE

SIDNEY N. PARKINSON, M.D., OAKLAND, CALIF.

While dermoid cysts in general are not rarities, one located on or near the dorsum of the nose seems to be unusual and of special interest. In reporting such a cyst in 1938, Hagens [1] stated that only 3 had been reported in American literature and but 26 in the whole literature on dermoid cysts.

Amelia C., a girl of 4, was brought to my office in January 1938. She presented a swelling of the nasal dorsum about the size and shape of a small almond, which was known to have been present at least two years. The mass

Fig. 1.—A section through the wall of the dermoid cyst, showing a sebaceous gland and hair follicles, with characteristic squamous cells. Numerous inclusions such as these were seen throughout the sections.

was soft and movable. It showed no evidence of inflammation and was not tender. Examination of the child revealed nothing otherwise remarkable.

Although the nature of the mass was uncertain, it was considered advisable to remove it if for no other reason than to correct the deformity. On January 24 removal was accomplished, through a linear incision along the dorsum of the nose. Considerable fibrous tissue was encountered, requiring sharp dissection throughout. A depression was noted, centered about one-third way down along the dorsum, and the nasal bones appeared to be shortened. On completion of

1. Hagens, E. W.: Congenital Dermoid Cyst and Fistula of the Dorsum of the Nose, Arch. Otolaryng. **28**:399 (Sept.) 1938.

dissection it was found that small openings had been made into the most anterior portions of both nasal fossae. No attempt was made to suture these separately. The wound was closed without drainage. Convalescence was uneventful. Massage of the nasal bridge for a moment each day was carried out for several weeks following healing, to prevent adherence of the scar.

Examination of the dissected mass by Dr. Paul Michael, pathologist, revealed a tangle of dark hair in a quantity of greasy putty-like material. The wall was thickened by fibrosis. The sac was lined by flat squamous epithelium,

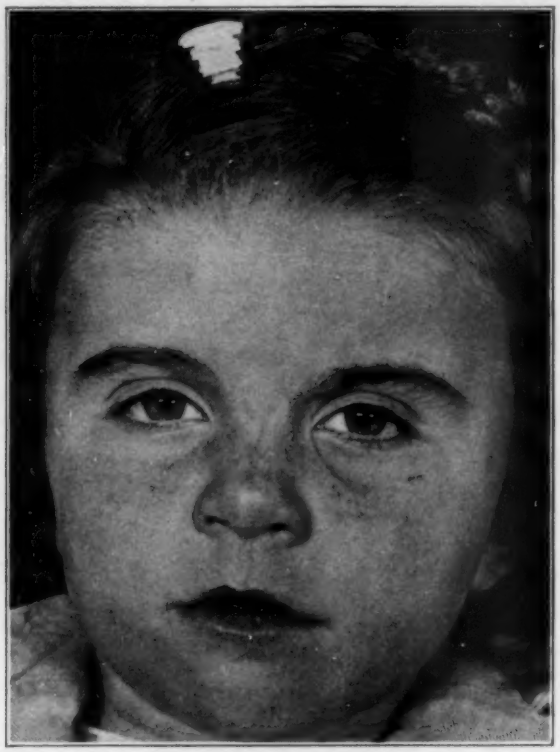

Fig. 2.—Amelia C., aged 7 years. A dermoid cyst of the nasal bridge was removed three years before the photograph was taken. The linear scar along the nasal dorsum is scarcely discernible. The nose is slightly broadened but otherwise presents no deformity.

with many hair follicles and sudoriferous and sebaceous glands in the surrounding wall.

The child was last seen Jan. 4, 1941, three years after the operation. The line of incision on the nasal dorsum was scarcely visible and was entirely nonadherent. The nasal bridge was slightly broad but showed no depression or other deformity.

Dermoid of the nasal bridge arises, as do dermoids elsewhere, from epidermal inclusions or sequestrations during embryonic development.

By the time the growth is clinically recognizable, there is commonly close association between the wall of the cyst and the nasal mucosa, due to contiguity. While instances of direct connection between the dermoid wall and the dura are not recorded in the literature, yet instances of close association due to extension toward the frontal lobe are mentioned. Secondary infection sometimes occurs, probably from trauma. Thus, a certain amount of hazard is associated with removal, and various suggestions have been made for the development of an effective yet safe surgical technic. Further comment and a complete bibliography are contained in Hagens' article,[1] to which the reader may refer.

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SUBCUTANEOUS EMPHYSEMA AFTER TRACHEOTOMY

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EDITED BY LOUIS E. SILCOX, M.D., PHILADELPHIA

Subcutaneous emphysema after tracheotomy is a well known entity. The 2 cases to be described in this paper may be of some interest, if only to demonstrate the difficulties of a specialist working under unfavorable circumstances.

REPORT OF CASES

CASE 1.—A 40 year old woman gave a history of hoarseness which started at 3 years of age and became progressively worse. One year before her case was brought to my attention, dyspnea developed, associated with attacks simulating asthma, which had become worse in the last three months. For two weeks the dyspnea had been so severe that she could not sleep, and she had suffered a heavy loss of weight. Several physicians had treated her for asthma. The first laryngoscopy was done at her home a month prior to my examination of her, and cancer of the larynx was diagnosed. She was sent to Tegucigalpa to consult an internist, Dr. E. Hirsch, who promptly called me because of the severe dyspnea and aphonia. On examination she was found to be moderately emaciated and showed great anxiety because of her difficulty in breathing. The examination of her chest revealed bronchitis, emphysema and tachycardia.

The external examination of the larynx and the neck revealed no abnormalities. The nose and the pharynx were normal. The laryngoscopic examination, which was brief because of the dyspnea, showed a large tumor with a nodular surface. The vocal cords could not be seen, but from the movements of the arytenoid cartilages it was assumed that they were not completely paralyzed.

The patient had come from San Pedro Sula by airplane a few hours before the examination. That town is 60 meters above sea level and has the typical hot climate of the tropics. Tegucigalpa, where the examination was made, is more than 3,000 feet (914 M.) above sea level and is comparatively cool. The possibility of variations in atmospheric pressure aggravating her condition was taken into consideration before a decision was made as to what course to follow. Time did not permit biopsy, serologic test or roentgen examination. Her condition was becoming steadily worse, and because we did not feel that she could stand a major procedure, an inferior tracheotomy was done, with the area under anesthesia induced by infiltration of a 1 per cent solution of procaine hydrochloride.

The patient was nearly unconscious when the operation was started. Little bleeding was encountered, and a cannula was inserted into the trachea. When this was done, her breathing stopped. She became cyanotic and pulseless. After artificial respiration and injections had been given, she started to breathe again. but her general condition was poor. The following day, subcutaneous emphysema of the head, arms and breasts developed; her head looked like a balloon. Her

breathing was difficult and her pulse rapid with frequent changes in quality. The position of the cannula was correct, and the inner cannula was cleared regularly. The emphysematous parts of the breasts and neck were pierced by hollow needles to drain the air; this procedure alleviated the pain. Her condition during the next few days was serious. She had a rapid pulse, and there was much coughing with expectoration of purulent sputum through the cannula. Roentgen examination showed extensive emphysema of the lungs and shadows about the size of a silver dollar in the lower lobe of each lung; these were diagnosed as abscesses or pneumonic foci. The Kahn test was negative.

She improved gradually in the following weeks. Her pulse rate became normal, and her breathing through the cannula easier. Expectoration stopped, the emphysema of the skin and lungs disappeared, and on fluoroscopic examination the lungs were observed to be clear. The general condition improved so much that a biopsy specimen was taken. Because of the inadequate laboratory facilities, the histologic study was not complete, but signs of malignancy could not be seen. There was no change in the laryngoscopic findings except that on one or two occasions the tumor was found below the vocal cords and the cords moved freely. It was assumed that the tumor was pedunculated and not malignant.

Four months after the tracheotomy, a thyrotomy was performed with the patient under local anesthesia. The thyroid cartilage was soft and cut with ease. There were hard papillomatous growths in the region of both vocal cords. There were more of these on the left side, where they were localized into a mass the size of a large hazelnut. Those on the right side were small. The tumor on the left had a pedicle and was removed by a Brüning tonsil snare. The base of the tumor was cauterized with an electric cautery. There was little bleeding. The interior of the thyroid cartilage was packed with iodoform gauze, and the patient continued to breathe through the cannula of the tracheotomy tube. After twelve hours the packing and cannula were removed, the edges of the wound were pressed together by a compression bandage, and the patient breathed freely through the normal passages. There were no complications, and the wound healed well except for a small fistula which was present when she left the hospital against advice. Her general condition was good, but she remained aphonic. The histologic diagnosis from the Gorgas Memorial Laboratory in Panama, Republic of Panama, was papilloma with no sign of malignancy.

Case 2.—A 16 year old girl complained of chronic nasal discharge, hoarseness and dyspnea, which had been getting progressively worse in the last few months. Rhinoscleroma had been diagnosed but not verified by bacteriologic and histologic examination. The Kahn test was negative.

Examination revealed a hard tumor about the size of a small hazelnut on the left side of the tip of the nose, fixed at the base. Both nostrils were closed by constricting scars, so that anterior rhinoscopy could not be done. Crusts and mucopurulent secretion were found at the entrance to the nares. There were broad scars on the posterior wall of the pharynx. The vocal cords were immovable in the midline; their median edges were of a reddish color.

Immediate tracheotomy seemed necessary because of the severe dyspnea. This was done at the General Hospital where only a few instruments were available; and the only cannula at hand was too small. Immediately after the operation and the insertion of this small cannula, subcutaneous emphysema developed which involved the head, neck and surrounding tissues. Treatment was carried on by another physician, and the outcome cannot be reported at this time.

COMMENT

In both cases the dyspnea had persisted for a long time. In the first case it was caused by a large papilloma of the larynx which had been practically untreated for nearly forty years. In the second case the cause of dyspnea was possibly scleroma of the larynx. In the first case emphysema of the lungs was proved present by roentgen examination, and in the second case it probably developed, although it was not demonstrated. In both cases there was extensive subcutaneous emphysema which developed after tracheotomy.

The commonest cause of subcutaneous emphysema after tracheotomy is slipping of the cannula because it is either too short or too small. This was probably the cause in the second case. Another cause may be obstruction of the inner cannula by secretion. This was probably not the cause in the first case, because the cannula was cleaned regularly despite the lack of trained personnel. Another cause may be that the tissues are made too loose when the trachea is opened. Wilms stated that subcutaneous emphysema cannot occur if the trachea is fixed by sharp hooks until the cannula has been introduced. Injury to the mucous membrane of the posterior wall of the trachea during the incision may be a factor in the production of emphysema. Although subcutaneous emphysema generally may not be a serious complication, it may prove to be in cases in which the heart and circulation are in poor condition, as in case 1. Piercing of the emphysematous parts with hollow needles seems to be of some help.

SUMMARY

Two cases of subcutaneous emphysema after tracheotomy are reported. In the first case the stenosis was caused by papilloma of the larynx, untreated for forty years, and in the second case the cause was possibly rhinoscleroma. The etiologic factors of emphysema are discussed.

INTRACRANIAL COMPLICATIONS FOLLOWING ACUTE EXACERBATION OF CHRONIC SINUSITIS

W. E. Grove, M.D., Milwaukee

J. T. M., a white man 30 years of age, entered the Milwaukee County Hospital on March 13, 1941, complaining of frontal headache, left orbital swelling and general malaise. The illness began one week before admission, when he contracted a sore throat and a head cold. Four days later a severe left frontal headache developed with a feeling of malaise. On the following day the left upper eyelid became swollen and the headache, localized to the left frontal area, became more severe. The patient had been nauseated and had vomited once before admission to the hospital.

His previous health had always been good except for frequent colds, intermittent attacks of sinusitis and frontal headaches.

Physical examination on admission revealed a white man 30 years of age, lying in bed with the left upper eyelid swollen shut and edema extending over the left side of the face and out toward the left temple. His temperature was 102 F.; his pulse rate, 116; his respiration rate, 24; his blood pressure, 130 systolic and 60 diastolic. The examination of the head and scalp revealed no abnormalities. The left upper eyelid had a red and tender swelling, extending to the ciliary ridge but not on to the forehead. The supraorbital ridge appeared roughened and showed increased induration to palpation over its middle portion. Laterally the edema extended over the parotid area. The right nasal passage was clear, and the right turbinates appeared normal. The left turbinates were red and swollen, and pus was seen to issue from the middle meatus. There was tenderness over the floor of the left frontal sinus and over its anterior wall. The rest of the physical examination yielded negative results.

On March 14 the patient was lethargic, though well orientated. His left upper eyelid was considerably swollen. The cellulitis had extended over the forehead to the hair line. The movements of the left eye seemed normal. A neurologic examination gave negative results. Roentgenographic examination revealed that the sinuses on the right side were clear but that the left antrum was cloudy. At 10:30 p. m. a trephine opening was made in the floor of the left frontal sinus and a rubber tube drain inserted. The sinus was filled with thick pus. At the same time and through the same superciliary incision a large abscess in the left upper lid and in the anterior portion of the left orbit was evacuated. By this time the induration and edema had extended upward above the hair line and backward on the left parietal bone almost as far back as the occiput.

From this time the course was downward. The patient became very restless and incoherent. The temperature, which had been septic, ranging up to 104 F., began to rise and reached 105.8 F. rectally at 4 p. m. on March 15. A spinal tap revealed a clear fluid under normal pressure. This fluid showed 25 white blood cells and 2 red blood cells; a blood examination made at this time showed 15,550 white blood cells, 4,450,000 red blood cells and a hemoglobin value of 91 per cent. The neutrophils were 96 per cent, of which 23 per cent were young forms. The patient died at 4:30 a. m. on March 16.

Autopsy (Drs. J. C. Grill and J. Kuzma).—The body was that of a well developed and well nourished white man, whose body length was 177 cm. and whose estimated weight was 185 pounds (83.9 Kg.).

The left side of the scalp presented rather pronounced swelling and pitting edema extending over the vertex and almost to the occiput. The left side of the forehead also was swollen and discolored. The left temporal area between the eye and the hair line was considerably swollen and marked with reddish discoloration, in which thrombosed vessels were easily palpable and readily visible as bluish cords.

The left eyelids were swollen. The left bulbar conjunctiva was edematous, and both scleras were icteric. The pupils were round, equal and medium sized.

The left supraorbital ridge was flattened. The left eyebrow was shaven, and from its medial portion, extending laterally, there was a surgical opening of 5 cm. This communicated with the frontal sinus, and at its medial angle a small rubber tube drain. From this area a heavy grayish brown foul-smelling material could be expressed. The nose and mouth were essentially normal.

Internal examination was limited to the head.

The scalp stripped rather easily. It showed on the left the aforementioned edema extending from the supraorbital region to the occipital region. There was also reddish patchy discoloration of the soft tissues of the scalp on the left. The left temporal muscle showed grayish discoloration in places and was markedly swollen; between it and the bone were pockets containing a thick purulent exudate. The calvarium was normal in thickness. At the left frontotemporal area there was noticeable grayish discoloration of the external table, but there was no detectable softening of the bone.

The dura stripped easily, and that over the left anterior fossa contained three patches covered by a heavy layer of purulent exudate. The largest of these measured 3 by 1 cm. On exploration of the sinuses of the head, the left frontal sinus was found to contain the material mentioned before and to have prominent vascular communication with the floor of the anterior fossa, in which the aforementioned epidural abscesses had been found. The ethmoid sinuses were quite large and were filled with a somewhat gelatinous material; their mucosa was congested but otherwise normal.

On removal of the hypophysis the floor of the hypophysial bed was also observed to be covered by a heavy purulent exudate. The underlying bone was soft and discolored grayish and was thin. On exposure of the sphenoid sinus there was an extremely putrid odor. The sinus cavity was filled with a heavy purulent exudate.

The brain weighed 1,400 Gm. The convolutions were flattened and swollen, and the leptomeninges were congested throughout. The vessels forming the circle of Willis were thin and elastic. On section of the brain the tip of the left frontal lobe contained a 1 cm. area of superficial cortical necrosis. Further horizontal and vertical sections of the brain revealed congested vascular markings and perivascular edema.

The anatomic diagnosis was: purulent inflammation of the left frontal and sphenoid sinuses; extradural abscesses in the left anterior fossa and about the body of the pituitary gland; superficial cortical necrosis in the left frontal lobe; congestion and edema of the leptomeninges and brain substance; cellulitis of the left eyelid and left side of the scalp; thrombophlebitis in the left temporal area; osteomyelitis of the left frontal bone and of the body of the sphenoid bone.

SUMMARY

A case of intracranial complications following acute exacerbation of chronic sinusitis is presented.

There were several interesting features: the neglect of the chronic sinusitis; the short fulminating course (only about ten days elapsed between the onset of the acute infection of the upper respiratory tract and death); the multiplicity of the complications (orbital abscess, osteomyelitis of the frontal bone and the body of the sphenoid, extradural abscesses in the anterior fossa and about the sella turcica, beginning meningitis, encephalitis and focal necrosis of the brain tissue in the frontal lobe and increased intracranial pressure); the route of the infection from the infected sinuses to the intracranial structures by vascular invasion through an osteomyelitic, but otherwise intact bone.

GLIOMA (ASTROCYTOMA) OF THE NARES

ALFRED A. SCHWARTZ, M.D., AND HAROLD J. ISAACS, M.D., NEW YORK.

Nasal tumors composed wholly or in part of brain tissue are rare. They were first described by Berger [1] 1890. In 1928 Eckert-Möbius,[2] in a review of the literature, could find only 11 recorded. Since then several others have been reported.

Such a growth may appear in any part of the nose and is in all cases congenital. It shows little or no tendency to grow, and there is no record of metastases in any instance. In some cases the growth has recurred after removal (probably incomplete), necessitating reoperation. According to site, there are three types, extranasal, intranasal and mixed intranasal and extranasal. In the majority of instances the growth has been of the extranasal type, the most frequent site being over the dorsum of the nose, immediately below the glabella. Here the growth takes the form of a firm elastic circumscribed mass, firmly attached to the skin and somewhat less firmly attached to the underlying nasal bones. The intranasal growth usually is somewhat softer and generally resembles a nasal polyp springing from the roof of the nose except that it is less translucent and of a firmer consistency. In the mixed type there is an external swelling, and part of the growth is intranasal.

Anatomically these cases fall into two groups. In one group there is a direct connection with the brain; in the other there is none. In the first group the growth is joined to the brain either by a connective tissue stalk or by neural tissue. In some of these cases a direct connection with the ventricles or the subarachnoid space has been demonstrated by the escape of cerebrospinal fluid from the pedicle, necessitating ligation. This connection usually passes through a dehiscence in the cribriform plate in the neighborhood of the foramen caecum. In the second group there is no communication between the brain and the tumor. The tumor, as far as it has been possible to demonstrate, is entirely independent.

Some authors (Berger,[1] Rocher and Anglade [3] and Guthrie and Dott [4]) regarded the tumors of these two groups as part of a series—encephalocele, encephaloma and glioma—all arising in the same way, as buds from the anterior cerebral vesicle, some retaining and others losing their central connection. Others believe that the growths may arise from local neural elements in the nares. The preponderance

1. Berger, P.: Considérations sur l'origine, le mode de développement et le traitement de certaines encéphalocèles, Rev. de chir. **10**:269, 1890.

2. Eckert-Möbius, A., in Denker, A., and Kahler, O.: Handbuch der Hals-Nasen-Ohren-Heilkunde, Berlin, Julius Springer, 1928, vol. 5, p. 153.

3. Rocher, H. L., and Anglade: Les fibriogliomes de la région nasale, Rev. de chir. **62**:147, 1924.

4. Guthrie, D., and Dott, N.: Occurrence of Brain Tissue Within the Nose: So-Called Nasal Glioma, J. Laryng. & Otol. **42**:733, 1927.

of opinion, however, is that the independent growths are not of the same type as those having central connections but are congenital embryonal rests of displaced neural tissue.

Zöllner [5] pointed out that not only may "congenital glioma" and encephalocele closely resemble each other in site and clinical appearance but they may show exactly the same histologic picture. Hence, biopsy alone may not sufficiently distinguish between them. The differentiation is important, because excision of an encephalocele may lead to meningitis.

The terms used by various authors to describe the histologic structure of these growths comprise "glioma" (Zöllner,[5] Guthrie and Dott,[4] Anglade and Philip,[6] Berblinger [7] and Clark [8]), "fibroglioma" (Rawson and Vivoli [9] and Rocher and Anglade [3]), "glioblastoma" (Eigler [10]), "encephaloma" (Browder [11] and Rocher [12]), "esthesioneuroblastoma" (Portmann, Bonnard and Moreau [13]) and "esthesioneurocytoma" (Berger and Coutard [14]). This multiplicity of terms is not surprising in view of the fact that the classification of tumors containing nerve tissue has up to the present caused considerable confusion. Bailey and Cushing [15] have urged that these tumors be classified morphologically, according to the predominating type of cell. Scherer [16] expressed the opinion that it is difficult to determine the predominating type of cell in all parts of the growth and urged classification on an embryologic basis. Ewing [17] stated the belief that it is premature, in view of present knowledge of the tumors, to urge adoption of a complex classification on this basis, because, if one judges from experience with other organs, it is conceivable that adult glial cells in new growth formations resemble certain embryonal cells. Such a resemblance would not signify either

5. Zöllner, F.: Angeborenes Gliom der Nase? Frankfurt. Ztschr. f. Path. **49**: 82, 1935.

6. Anglade, A., and Philip: Le gliome des fosses nasales, Cong. franç. d'oto-rhino-laryng. **8**:32, 1920.

7. Berblinger, W.: Gliom von seltener Lokalisation, Zentralbl. f. allg. Path. u. path. Anat. **31**:201, 1920.

8. Clark, J. P.: Glioma of the Nose, Am. J. M. Sc. **129**:769, 1905.

9. Rawson, J. D., and Vivoli, D.: Los fibriogliomas de la región nasal, Rev. méd. latino-am. **14**:860, 1929.

10. Eigler, G.: Drei seltene Geschwülste der oberen Luftwege, Hals-, Nasen- u. Ohrenarzt (pt. 1) **28**:320, 1937.

11. Browder, J.: Encephaloma or So-Called Nasal Glioma, Ann. Otol., Rhin. & Laryng. **38**:395, 1929.

12. Rocher: Un cas d'encéphalome, Bull. Soc. d'anat. et de physiol. de Bordeaux **23**:234, 1902.

13. Portmann, G.; Bonnard, and Moreau: Sur un cas de tumeur nerveuse des fosses nasales esthésioneuroblastome, Acta oto-laryng. **13**:53, 1928.

14. Berger, L., and Coutard, H.: L'esthésioneurocytome olfactif, Bull. Assoc. franç. p. l'étude du cancer **15**:404, 1926.

15. Bailey, P., and Cushing, H.: A Classification of the Tumors of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.

16. Scherer, H. J.: Critical Review: The Pathology of Cerebral Gliomas, J. Neurol. & Psychiat. **3**:147, 1940.

17. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 424.

identity with or origin from embryonal cells. He expressed the belief that for the present it is wise to accept the classification proposed by Bailey and Cushing.[15]

These authors in a study of 378 tumors of the brain classified the more common tumors as follows:

1. Astrocytoma	136
2. Glioblastoma multiforme	117
3. Medulloblastoma	55
4. Astroblastoma	20

The other groups are much smaller and are omitted here.

As will be noted, the most common form is the astrocytoma. This is the so-called classic glioma. The predomniating cell is star shaped, with an eccentric nucleus and fibrillary processes. The protoplasmatic astrocytoma contains a preponderance of cytoplasmic astrocytes, the fibrillary astrocytoma a preponderance of fibrillary processes. The protoplasmatic astrocytoma, slightly more active, occurs most frequently in the middle or deeper layer of the cerebral cortex; the fibrillary astrocytoma occurs more often in the subcortical regions. Astrocytomas grow slowly, mitotic figures are rarely seen and, according to Bailey and Cushing, there is no record of a cerebral glioma having given origin to metastases.

In view of the great number of terms used in describing these nasal neoplasms of neural origin it is not surprising that in the majority of the cases reported it is impossible to determine just what type of tumor was present. It is probable, however, that if Bailey's classification had been used, the majority of the tumors would have been considered to be astrocytomas.

REPORT OF A CASE

E. N., a boy aged 13 months, was first examined on Feb. 28, 1940. At birth a mass was noticed in the vestibule of the right naris. According to the mother, this had been slowly growing larger and now completely blocked the right nasal orifice. The child kept his mouth open, especially at night, and his sleep was restless. There had been occasional bleeding from the growth, described as a slight ooze.

Examination showed a mass the size of a small bean, elongated and rather firm, arising from the right lateral nasal wall at the mucocutaneous junction and extending upward and backward along the lateral nasal wall toward the right inferior turbinate. It completely occluded the right naris. The posterior limit of the tumor could not be determined at this examination.

On March 1 the growth was removed at Beth Israel Hospital. It consisted of two parts. The anterior portion, attached to the mucocutaneous junction, was rather firm. The posterior portion was more polyp-like and translucent. It is probable that both were parts of one mass and not two separate tumors. After the growth had been completely removed with snare and punch forceps, the base was thoroughly curetted. The interior of the right naris could then be visualized and was seen to be entirely clear of tumor.

The patient was last examined on Jan. 15, 1941, ten and one-half months after operation. There had been no recurrence of the tumor. The removal of the nasal obstruction was followed by a marked improvement in the general condition.

The nature of the tumor was determined first by Dr. Alfred Plaut, pathologist of Beth Israel Hospital, and then by Dr. Charles Davison, neuropathologist of Montefiore Hospital for Chronic Diseases and consultant neuropathologist of Beth

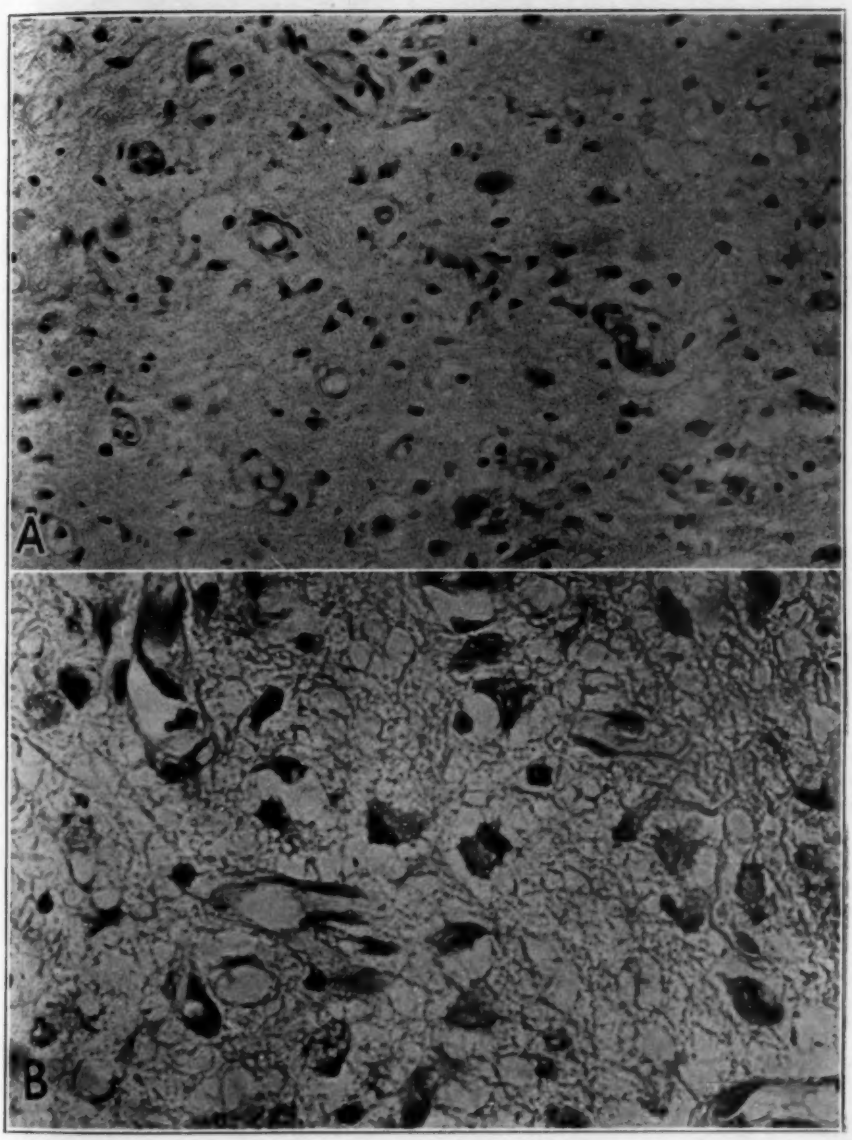

Fig. 1.—Protoplasmatic and fibrillary astrocytes; cresyl violet; *A* × 200; *B* × 400.

Israel Hospital. Dr. Davison's report is quoted in the following paragraphs. The photomicrographs were taken by him in the neuropathologic laboratories of Montefiore Hospital for Chronic Diseases.

Microscopic Examination.—Slides of sections from the tumor of the right nostril were stained by the cresyl violet method.

The tumor consisted of an outer layer of epithelial tissue, which was about normal in appearance. In the subcutaneous tissue were numerous vascular chan-

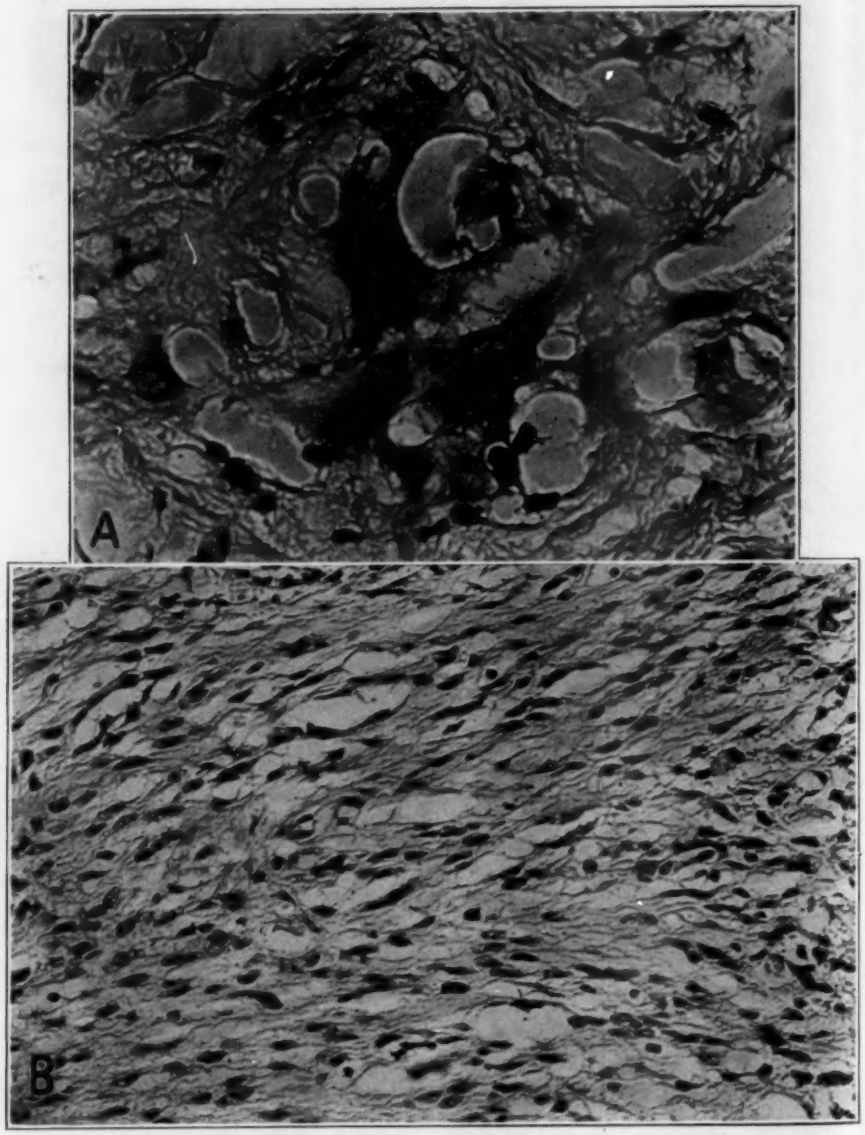

Fig. 2.—*A*, several multinucleated protoplasmatic astrocytes; cresyl violet; × 400. *B*, section of the tumor, showing its fibrillary character; cresyl violet; × 200. Notice the parallel arrangement of the cells.

nels. Between these were large pale-staining cells, with a vesicular nucleus usually displaced at the periphery; some of these cells had various processes which were

identified as glial fibers. Many of them resembled either gemästete glia cells or large protoplasmatic astrocytes. Some of the cells were multinucleated. Others were elongated and had the appearance of spongioblasts; these had tremendous processes which extended into the matrix. The intervening tissue contained irregular elongated nuclei, which had a slight palisade arrangement and looked more like fibroblasts and endothelial and mesenchymal cells. Ganglion cells were not present. In other sections the tumor consisted of numerous capillaries lined by single layers of endothelial cells; between these were numerous oval and irregular darkly stained nuclei.

The microscopic diagnosis was protoplasmatic astrocytoma.

Comment.—It is possible that this tumor arose from the neural elements belonging to the subcutaneous tissue or from neural embryonal rests.

7 West Seventy-First Street.

897 Park Avenue.

Clinical Notes; New Instruments and Technics

A BRONCHOSCOPIC OBSERVATION IN CASES OF PLEURAL EFFUSION

JOEL J. PRESSMAN, M.D., LOS ANGELES

For a number of years I have from time to time noticed that in certain bronchoscopic examinations the carina, or spur dividing the left from the right main stem bronchus, underwent unusually wide to and fro excursions in a lateral direction. Such movements were always synchronous with cardiac pulsations and closely resembled the multiphasic excursions of an electrocardiographic string during recording of the heart beat. Sometimes these carinal excursions were very wide, greater than the width of the lumen of a 7 mm. bronchoscope.

At first I paid little attention to this phenomenon, simply considering it to be an exaggerated transmitted impulse of the heart beat or of the great vessels of the thorax, and attached no particular significance to it. More recently I have almost come to the conclusion that such a transmitted impulse, which reflects itself in the usually wide movement of the carina just described, takes place only in cases of pleural effusion. The widest excursions were found in cases of massive effusion on the left side, but they have also occurred to some extent in cases of effusion on the right. In some instances the amount of fluid present was not very great. The pulsatory movements differed from normal in that they were far wider than would normally be expected even allowing for physiologic exaggerations.

The explanation of this phenomenon is probably very simple. Physicians are all familiar, in cases of ascites, with the physical sign of so-called "fluid wave" or "fluid thrill" elicited by tapping one side of the abdomen with the hand while palpating the opposite side with the other hand. In the presence of abdominal fluid a transmitted impulse is felt by the palpating hand. This is due to the movement of fluid as a result of the tapping. Essentially the same thing occurs in the presence of pleural effusions except that the pulsating heart replaces the tapping hand and the impulse is conveyed to the main stem bronchi instead of to the palpating hand. This transmission of fluid movement to the bronchial tree manifests itself by carinal deviations which reflect, through the medium of fluid transmission, each pulsation of the heart, the auricular and ventricular beats separately, wherein lies the observed similarity to the movements of the electrocardiograph string.

It may be suggested that exaggerated reflections of cardiac pulsations can be observed in patients with other conditions as well as in those with pleural effusions. I have not, however, noticed any wide deviation of the carina in normal persons, although there is often a relatively minor pulsatile excursion. From time to time slight fluctuations can be discerned, but these are never very marked and do not resemble even remotely the wide fluctuations which take place in certain instances of pleural effusions.

This bronchoscopic sign has never been brought to my attention in cases of solid tumors of the mediastinum. It might, however, reasonably be expected that transmission of the beat of large mediastinal vessels to any intrathoracic structure, including the carina, may be very pronounced in any pathologic state producing a high pulse pressure, as, for example, aortic regurgitation or thyrotoxicosis. This may well be, but since I have always considered such diseases, especially if severe, contraindications to bronchoscopic examination, my bronchoscopic experience in their presence is extremely limited, so that I can neither support nor refute this possibility. Such diseases, however, are easily diagnosed, and in their presence it might well be that this bronchoscopic sign would not be of differential diagnostic value in so far as pleural effusions are concerned. Further observations are necessary to clear this point.

Much the same situation holds true for thoracic aneurysms. I imagine that a large aneurysm near the bifurcation of the trachea might produce a tremendous transmission of its pulsation to the carina, but I wonder if it would have the same biphasic character denoting auricular and ventricular beats as characterizes the carinal fluctuations due to pleural effusion.

Recently it has been necessary for me to examine bronchoscopically a patient with extreme hypertension. I looked especially for this sign but did not find it.

In those instances in which exaggerated carinal fluctuations have been present as a result of pleural effusion, there has almost always been noticed a marked reflection of the cardiac beat in the left main stem bronchus, as well as in the carina, but I have noticed bronchial excursion on numerous other occasions when there was no fluid, so that it has practically no differential diagnostic value in contrast to the same observations in the carina itself.

The practical application of this observation is perhaps limited. It would be of great value were it present in cases of small or localized effusions in which the clinical diagnosis is uncertain. In view of the limited number of observations that have been made, it must be considered unsafe to rule out the possibility of the presence of fluid if carinal fluctuations are absent; but in cases in which the diagnosis is in doubt between, let us say, solid tumors and mediastinal fluid, the presence of such exaggerated fluctuations would point strongly toward fluid. In cases of massive effusions, especially on the left side, the sign is almost always present, but in these instances the diagnosis is usually easily determined by other means, so that this bronchoscopic finding adds little or nothing of diagnostic value.

In a few isolated instances the practical application of this observation has been very helpful, for instance, in a case of encapsulated empyema in which pus was not obtained on repeated thoracenteses and further attempts had been abandoned. In this instance the presence of exaggerated carinal fluctuations led to further repeated chest taps with a trocar of very large caliber and ultimately to the successful withdrawal of extremely thick pus.

The purpose of presenting this observation prior to studying it in sufficiently great detail to warrant more definite statements is so that others will look for it too, under a great variety of circumstances. In any one clinic with limited material it is impossible to arrive at definite and final conclusions in matters of this sort. Further reports by other physicians will therefore be deeply appreciated.

1917 Wilshire Boulevard.

COMBINED SELF-RETAINING RETRACTOR FOR MASTOIDECTOMY AND EXTERNAL FRONTOETHMOIDECTOMY

WAYNE Y. H. HO, M.D., SHANGHAI, CHINA

Self-retaining retractors of various kinds are used in otolaryngologic operations. In brief, they are the self-retaining retractors for mastoidectomy and those for external frontoethmoidectomy. Each kind seems to have its own usefulness and merit.

The self-retaining retractor presented here is a combined, interchangeable, detachable and reversible one for use in mastoidectomy and in external fronto-

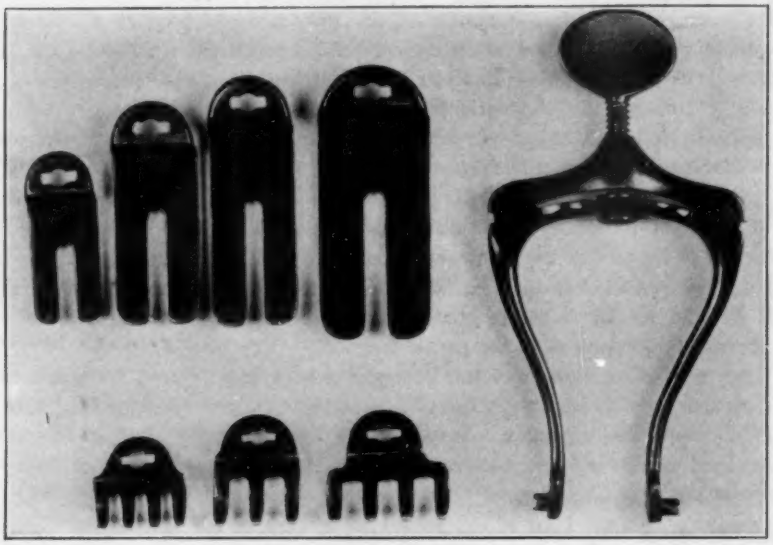

Fig. 1.—Combined self-retaining retractor for mastoidectomy and external frontoethmoidectomy.

ethmoidectomy. It consists of two parts: (1) a spreader with French lock joints on the legs and (2) blades with prongs of various sizes and types for specific operations (fig. 1). As a retractor for mastoidectomy, each leg of the spreader is fitted with a blade of three, four or more prongs to suit the surgeon's needs (fig. 2*A*). For external frontoethmoidectomy, the slotted blade desired for the retraction and protection of the orbital contents is attached to one leg of the spreader, and the blade with four prongs is fitted to the other for use on the nasal side (fig. 2*B*).

The combined self-retaining retractor described was constructed for me by V. Mueller & Company, Chicago.

From the Otolaryngologic Service of Dr. Lyman Richards, Department of Surgery, Peter Bent Brigham Hospital, and from the Otolaryngologic Service of the Children's Hospital, Boston.

This retractor is identical with McCullough's in its use in external frontoethmoidectomy, but it withstands strain in retraction better; for the pivot joint of the McCullough retractor to which the blade with four prongs is firmly fastened and nondetachable is replaced by the stronger, yet more serviceable French lock joint modification.

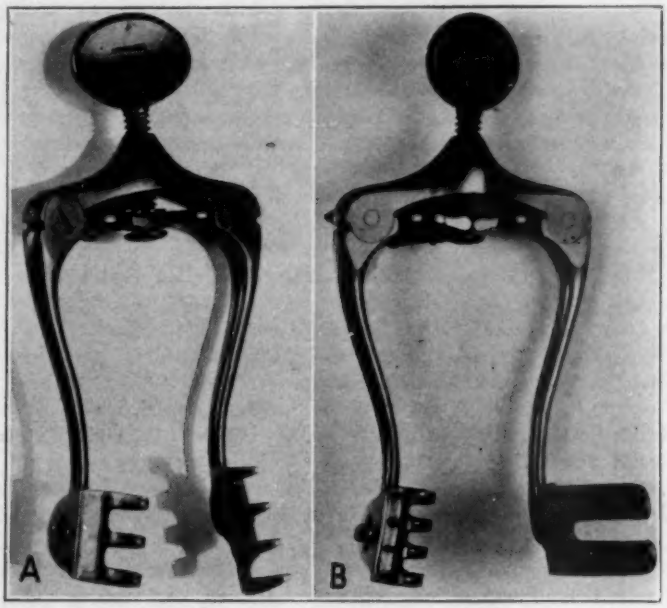

Fig. 2.—*A*, self-retaining retractor adjusted for mastoidectomy; *B*, the same retractor adjusted for external frontoethmoidectomy.

A SUBMUCOUS ELEVATOR

MYRON METZENBAUM, M.D., CLEVELAND

The Freer elevator is accepted as an excellent instrument. The shaft, which is held between the thumb and two fingers, is too thin to insure a firm grip; therefore the instrument may rotate or slip. The blunt separator with handle has a standard Freer separating end mounted in a hollow handle which fits well in

The blunt separator with handle.

the palm of the hand and is therefore less likely either to slip or to rotate. It is well balanced; so the surgeon has a sense of what is going on at the operating end. The instrument can be used as a regular submucous separator and is especially well adapted for the elevation of tissues in oral surgery. The instrument is available in 6 and 7 inch (15 and 18 cm.) lengths and is manufactured by the George Pilling Company, Philadelphia.

News and Comment

ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES

The annual meeting of the Association of Military Surgeons of the United States will be held Oct. 29 to Nov. 1, 1941, at the Brown Hotel, Louisville, Ky. All members of the medical profession are invited to attend as guests, particularly members of the medical defense committees.

War medicine and surgery have changed considerably since the previous emergency. Mechanization of armies and air bombardments have created new and difficult problems in traumatic surgery and methods of treatments of wounds and extreme abrasions. The session will conclude with a mass review of military medicine and an inspection of Fort Knox.

The preliminary program of professional sessions of the meeting follows:

Col. Harold D. Corbusier, Medical Reserve, United States Army: Presidential Address.

Rear Adm. Ross T. McIntire, Surgeon General of the United States Navy: "Policies and Activities of the Medical Department of the United States Navy in the Present National Emergency."

Dr. Warren F. Draper, Assistant to the Surgeon General, United States Public Health Service: "Present Policies and Activities of the United States Public Health Service."

Brig. Gen. Frank T. Hines, Administrator of Veterans' Affairs: "Progress of the Part the Veterans' Administration Is Playing in the National Defense Program."

Major Frank B. Wakeman, Medical Corps, United States Army, representing Major Gen. James C. Magee, Surgeon General, United States Army: "Present Policies and Activities of the Medical Department of the United States Army."

Col. Robert H. Duenner, Medical Corps, United States Army, Fort Knox, Ky.: "Medical Service of the Mechanized Forces."

Col. Paul E. Howe, Sanitary Corps, United States Army: "Nutritional Problems of the Army."

Col. Fred H. Albee, Medical Reserve, United States Army: "Treatment of Ununited Fractures of Importance to the Military Service."

Capt. Lucius W. Johnson, Medical Corps, United States Navy: "Medical Service at Remote Naval Bases."

Capt. William L. Mann Jr., Medical Corps, United States Navy: "Medical Arrangements for Combined Operations of Land Forces and Sea Forces" (with motion pictures).

Papers are scheduled from the following speakers, but the titles are not yet available:

Major Gen. C. R. Reynolds, United States Army Ret.

Brig. Gen. Leigh C. Fairbank, Assistant to the Surgeon General, United States Army.

Col. Irvin Abell, of Louisville, Ky.

Col. Leonard C. Rowntree, Medical Reserve, United States Army; Chief, Medical Division, Selective Service System, Washington, D. C.

Col. Raymond A. Kelser, Veterinary Corps, United States Army.

Lieut. Col. David N. W. Grant, Medical Corps, United States Army, Office of the Chief of the Air Corps.

Abstracts from Current Literature

Ear

SARCOIDOSIS OF THE EXTERNAL EAR. DAVID L. POE, Ann. Otol., Rhin. & Laryng. **49**:771 (Sept.) 1940.

Sarcoid of Boeck has been known for several decades and for long was thought to be a disease of the skin only. Today it is more fully recognized as a systemic ailment. Every organ of the body has been known to be affected, but little has been said about the ear. The cause of the disease is still unknown. The nodules are firm, nontender, noncaseating and of various sizes. Histologically the tissue resembles the small cell sarcoma. Many accept the disease as a type of tuberculosis, the cause of which is unknown. So far no satisfactory therapy has been found.

Poe reports a case of sarcoidosis in a Negress of 38 years, in whom the first symptoms apparently had been present twenty years before. When she was seen, the condition was general throughout the body. He describes the findings in some detail and expresses the belief that this is the first case in which involvement of the external ear has been described.

M. V. MILLER, Philadelphia.

GEOGRAPHIC VIRULENCE OF MASTOIDITIS. MERCER G. LYNCH, J. A. M. A. **115**:826 (Sept. 7) 1940.

Lynch, through the cooperation of physicians in other cities, conducted a series of studies by taking cultures of hemolytic streptococci from various parts of the country, transplanting these cultures to blood agar and then making standard solutions so that all solutions for experimental and testing purposes were of equal strength.

The solutions were subjected to various temperatures, such as are found in different localities, as well as to sudden temperature changes, and used on guinea pigs.

It was found that when the temperatures remained uniform the organisms were less virulent and when the temperatures varied or presented sudden changes, the virulence of the organisms increased.

Since every variety of climate and weather condition is found within the United States, the fluctuating virulence of the organisms can be used to explain the clinical reports that the highest incidence of mastoiditis is in March, after a gradual rise in December and January, with a decline after March to a low point in August. Since the last month is the time of most uniform temperature, the virulence of the organisms is then lessened.

In general it may be said that the incidence of mastoiditis is greater in the northern parts of the country and less in the southern parts. Climates vary—persons vary—and so do organisms in response to environmental changes.

GORDON, Philadelphia.

THE TREATMENT OF CHRONIC DEAFNESS AND TINNITUS AURIUM WITH PROSTIGMINE. KARL M. HOUSER, EDWARD H. CAMPBELL and H. SCHLUEDERBERG, J. A. M. A. **115**:994 (Sept. 21) 1940.

The authors report the results in a series of 56 patients with chronic deafness studied and treated in the clinics of the University of Pennsylvania School of Medicine, the Pennsylvania Hospital and the Hospital of the Protestant Episcopal Church. Prostigmine methylsulfate was given hypodermically, along with the oral administration of prostigmine bromide. Local treatment of the eustachian

tube, the nose and the throat was continued and was not interrupted during the administration of prostigmine methylsulfate.

Careful tests of the hearing were made at intervals, audiograms were taken every two or three weeks and tuning fork tests were also made. During the treatment period 1 cc. of prostigmine methylsulfate (1:2,000 dilution) was given hypodermically every third day, and, in addition, 15 mg. of prostigmine bromide was given after meals during the first six weeks of treatment.

The authors wisely point out that "It is well recognized by otologists that audiograms taken on separate occasions will not always give identical readings in either the normal or the hard of hearing patient . . . one allows for a +7 or —7 decibel variation. This fact should be understood lest one draw inaccurate conclusions from a decibel change of 15 or less."

Their conclusions are as follows:

"1. In a series of fifty-six carefully tested and controlled cases of chronic deafness, prostigmine treatment failed to exert a definite beneficial effect.

"2. It is doubtful that any real effect is produced on tinnitus aurium. Seven patients felt that some improvement had taken place. None lost their tinnitus completely.

"3. Our studies lead us to believe that the treatment of deafness and tinnitus aurium with prostigmine is of but little if any value." GORDON, Philadelphia.

OTITIC MENINGITIS OF LABYRINTHINE ORIGIN. CARLOS P. MERCADINO and LUIS A SAMENGO, Rev. med. y cien. afines **2**:863 (Nov. 30) 1940.

Mercadino and a Samengo report a case of chronic suppurative otitis media with cholesteatoma of four years' duration. One week before admission of the patient to the hospital acute coryza developed, which was followed by labyrinthitis and meningitis. Although there was a marked increase of spinal fluid pressure and an uncountable number of cells within the fluid, with a preponderance of polymorphonuclears (87 per cent), the culture of the fluid was sterile.

A radical mastoidectomy was performed, with a labyrinthectomy, according to the Neumann technic. There was immediate postoperative facial paralysis, which cleared up in a few days. Treatment was supplemented by oral and parenteral administration of sulfanilamide, and the patient made an uneventful and complete recovery. PERSKY, Philadelphia.

Larynx

CHRONIC HOARSENESS: REPORT OF THREE HUNDRED CONSECUTIVE CASES. J. C. DAMITZ and J. LEWIS DILL, Ann. Otol., Rhin. & Laryng. **49**:996 (Dec.) 1940.

Chronic hoarseness is not a clinical entity but a symptom of some condition preventing normal functioning of the larynx. The authors analyze their cases and point out that the period from the onset of hoarseness to proper examination and diagnosis varied from over two months to over two years and that the symptom is often neglected not only by the patient but frequently by the physician. Direct or indirect laryngeal examination, or both, should be made on all patients. Whenever indicated, bacteriologic studies of the larynx, a physical examination of the patient, including complete blood cell counts and a chemical examination of the blood, Wassermann tests of the blood and spinal fluid, tuberculin studies, bronchoscopic and esophagoscopic studies and biopsies should be made. In 67 per cent of the 300 cases analyzed the hoarseness was due to nonspecific laryngitis and paralysis of the vocal cords, about equally divided. There were 41 benign to 25 malignant growths. Carcinoma was noted in 25 cases, tuberculosis in 23 and syphilis in only 1. Early, thorough and repeated examination of the larynx in cases of chronic hoarseness is urged and emphasized.

M. V. MILLER, Philadelphia.

Nose

OSTEOMYELITIS OF THE FRONTAL BONE AS A COMPLICATION OF FRONTAL SINUSITIS. H. P. MOSHER, J. A. M. A. **115**:1179 (Oct. 5) 1940.

Dr. Harris P. Mosher, dean of American otolaryngology, presents a full discussion of the great problem of osteomyelitis of the frontal bone. To attempt to abstract it would necessitate a revision of the text matter, for with his characteristic thoroughness Mosher has cleverly considered and pondered every angle, bringing his conclusions down to date forcibly and indelibly.

He warns of the pitting edema of the skin of the forehead (Pott's puffy tumor) as the first sign of involvement of the frontal bone, adding that microscopic specimens of removed bone have shown that the limit of the edema is a practical guide and, in fact, the only guide to the limit of infection in the bone marrow. Roentgenograms are seven to ten days late in providing positive evidence of osteomyelitis, because it takes that length of time for necrosis of bone to occur. Microscopic examination has shown that around an area of necrosis the infection extends radially for from 1½ to 2 inches (4 to 5 cm.).

Numerous details are carefully considered. The infection rapidly extends to the intracranial structures. It is important to remove the entire face of the frontal bone in one piece, starting the operation in clean bone. Modern plastic surgery can correct 90 per cent of the deformities.

Mosher strongly opposes installment operating in a case of fulminating osteomyelitis, and concerning this he says: "I should be willing to vote an antemortem halo to the man who can pick out at the outset and be right 75 per cent of the time which case will localize and not present complications."

I would advise every physician to read, digest and assimilate Mosher's scholarly presentation.

GORDON, Philadelphia.

OBSERVATIONS ON THE EPIDEMIOLOGY OF THE COMMON COLD. WILSON G. SMILLIE, New England J. Med. **223**:651 (Oct. 24) 1940.

Smillie calls the common cold a paradox and reiterates that often what is considered to be a common cold is one or another of a group of diseases presenting a similar syndrome. The similarity to the common cold of the symptoms of contagion at onset, of recurrent sinusitis, of transient allergy and of local mechanical irritation is cited. The true common cold is defined as a highly contagious disease entity, due to a filtrable virus having an incubation period of twenty-four to thirty-six hours, when it is most transmissible. It subsides in four to five days. The virus disease influenza is necessarily included in the discussion of the common cold, because the acute epidemic respiratory infections are not a single disease entity. An epidemic of respiratory infection at Northwest River, Labrador, known to be due to a single obvious origin, produced symptoms of all gradations. It is suggested that all acute epidemic respiratory infections are due to a group of closely related infectious agents.

A specific virus vaccine cannot produce more than the temporary immunity conferred by an attack of the disease itself. The serious consequences are due to secondary invasion by the prevailing organisms, most often the pneumococcus, quiescent before a cold. Theoretically, a vaccine prepared from the bacterial organisms most prevalent in the nasopharynxes of the members of a community should give some degree of protection against the most serious consequences of the common cold.

SCHALL, Boston.

Miscellaneous

SCARLET FEVER THERAPY: A COMPARISON OF CONVALESCENT SERUM AND SULFANILAMIDE. M. FOX and M. HARDGROVE, Am. J. M. Sc. **199**:495 (April) 1940.

The authors divide 300 hospital patients with scarlet fever into three groups: 100 with mild infection receiving nonspecific therapy and considered as controls,

100 with mild infection and moderately ill who were treated with azosulfamide (disodium-4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate) and 100 moderately or severely ill patients treated with convalescent serum. Among their observations were the following: The total number of complications was less in the serum-treated group; otitis media occurred eight times in the azosulfamide-treated group as against twelve times in the controls and eight times in the serum-treated group; cervical adenitis occurred eighteen times in the control group, fourteen times in the azosulfamide-treated group and eight times in the serum-treated group; no cellulitis or infection of an extremity occurred after the use of azosulfamide as against five such complications in the controls and four in the serum-treated group.

The authors feel that azosulfamide has a place in the treatment of scarlet fever. However, it is not for the toxic phase or type of the disease, which is best treated with convalescent serum. Four patients with hemolytic infections of the blood stream occurring in the course of scarlet fever received azosulfamide and recovered. Patients with otitis media and mastoiditis appear to have been helped by this drug. Two patients with hemolytic streptococcic meningitis following scarlet fever have been treated successfully with it. Improvement has also been noted after the use of azosulfamide in deep-seated recesses of infection, where surgical drainage necessarily complements the action of the drug.

GEORGE E. SHAMBAUGH JR. and MILTON L. JENNES, Chicago.

FOCI OF INFECTION IN PSYCHIATRIC PATIENTS. C. H. BROWN, Am. J. M. Sc. **199**:539 (April) 1940.

Brown made a study to determine the incidence of foci of infection in a group of patients who were apparently physically well. He chose a group of 239 psychotic patients, hospitalized because of mental rather than physical sickness. These patients were carefully examined for foci of infection by the proper specialists. The incidence of foci of infection for the entire group was 62.8 per cent. It is significant, however, that none of them had arthritis, and only 2 had rheumatic heart disease. Pemberton is quoted as having found an incidence of focal infection of 70 per cent in 1,100 patients with arthritis. Therefore, it appears that the incidence of focal infection is approximately the same in a group of patients with arthritis as in a group of apparently physically normal persons. It is therefore evident that more abnormality must be present in the patient with arthritis than just a focus of infection.

The most common focal infection was found to be nasopharyngeal (44 per cent), followed in order of frequency by dental (37 per cent), gynecologic (22 per cent), urologic (17 per cent) and cardiorespiratory infection (1.6 per cent). The two most common causes of focal infection in the nose and throat were chronic tonsillitis and sinus infection, found in 22 and 20 per cent, respectively, of these patients. The maxillary sinus was involved in 75 per cent of those showing sinus infection; the ethmoid labyrinth, in slightly under 25 per cent.

GEORGE E. SHAMBAUGH JR. and MILTON L. JENNES, Chicago.

VITAMIN A STATUS OF FAMILIES IN WIDELY DIFFERENT ECONOMIC LEVELS. P. B. MACK and A. P. SANDERS, Am. J. M. Sc. **199**:686 (May) 1940.

In a study of 100 families with widely varying incomes there was found to be a relation between the consumption of vitamin A and the response to the photometer test. These were in turn related to the income, the money spent for food and the educational level of the adult members of the family. Good average responses to the photometer tests were associated with uniformly high intakes of vitamin A. No family receiving under 8,000 U. S. P. units was in the highest class with respect to the bright light test, and few families receiving less than this amount were in the best class on the basis of the darkness regeneration test. Although a few families showing poorer response to the darkness adaptation tests

were receiving 12,000 U. S. P. units of vitamin A or more per person daily, the greater majority were receiving less, and almost half were receiving under 8,000 units.

GEORGE E. SHAMBAUGH JR. and MILTON L. JENNES, Chicago.

BRONCHIECTASIS: A STUDY OF PROGNOSIS BASED ON A FOLLOW-UP OF FOUR HUNDRED PATIENTS. KENNETH M. A. PERRY and DONALD S. KING, Am. Rev. Tuberc. **41**:531 (May) 1940.

The recent advances in the surgical treatment of bronchiectasis have suggested a wider application of this form of treatment. A study of the natural course of the disease was made in order to decide whether and in what circumstances a patient should be advised to submit to lobectomy, with its 10 per cent or less of operative risk.

A careful follow-up of 400 patients disclosed that in 69 per cent of all cases the disease had its onset in the first two decades of life, in 42 per cent, in the first, and in 27 per cent, in the second decade. This is interesting from the standpoint of etiology, since the onset of the disease is usually associated with such acute infections of the respiratory tract as are especially common in early life. Bronchiectasis is the result of the superimposing of an infection on a mechanical deformity, obstruction and dilatation, of the bronchi. Clinical and experimental observations have confirmed the view that the abnormal forces created by collapse of the lung are those most likely to cause dilatation of the bronchi. The frequency with which collapse occurs as a result of pneumonia, whooping cough, measles and other common diseases probably accounts for the high percentage of onset during the first two decades of life. Bronchiectasis does not result immediately on the development of the mechanical factors, since these conditions may lie dormant for many years, until purulent bronchitis is superimposed. The relation of sinusitis to bronchiectasis has been widely discussed but Perry and King are of the opinion that there is no causal relation between the two conditions.

Dilatation of the bronchi alone does not give rise to symptoms (except hemoptysis) until infection occurs. After infection, cough and purulent sputum occur, and hemorrhage in varying amounts is frequent. Hemorrhage developed in 148 of the patients studied; it was sufficient to cause death in 4 cases.

The most frequent complications were recurring attacks of apical pneumonia; less often there are observed pulmonary abscess, abscess of the brain, failure of the right side of the heart, empyema and pyopneumothorax.

A twelve year study of this group showed the mortality among the conservatively treated patients to be 26 per cent. In 116 cases the operative mortality from 122 lobectomies of the modern type was 3.3 per cent. From these observations it appears that lobectomy is the treatment of choice for bronchiectasis, especially for the unilateral condition.

EUGENE H. SMITH, Ogden, Utah. [AM. J. DIS. CHILD.]

ALLERGY OF THE UPPER AND LOWER RESPIRATORY TRACTS IN CHILDREN. FRENCH K. HANSEL, Ann. Otol., Rhin. & Laryng. **49**:578 (Sept.) 1940.

The paper is based on a study of 455 cases encountered in the pediatric allergy clinic of Washington University, St. Louis. This study consisted of a careful analysis of the nasal symptoms of sneezing, itching and obstruction, of gross nasal changes, of the cells of the nasal secretions, of roentgen findings and of bacteriologic findings. It was felt that of all these diagnostic factors the eosinophilia of the nasal and sinus secretions was the most positive sign of allergy. By cytologic study it was also possible to determine the clinical course of the disease in relation to acute, subacute and chronic complicating infection.

Hansel states that, although tonsillectomy is indicated in certain patients who have nasal allergy, it is important to bear in mind that the operation will not materially influence the allergic symptoms except in a small percentage of cases. The indications for tonsillectomy should be the same in children with nasal allergy

as in those without nasal allergy, but in any event the operation should be done only after allergic study.

Because edema of the nasal mucosa is commonly associated with nasal allergy, positive roentgen findings are the rule rather than the exception. Roentgenograms should not be taken during a period of acute exacerbation or during an acute or subacute infection, as the findings will be more pronounced than during the quiescent stage. Hansel feels that the studies indicated that sinus infection is not a common complication of respiratory allergy. He feels, too, that in the reports on sinal disease in children allergy is not given proper consideration as an etiologic factor and that a clear general understanding of sinal disease in children will not come until this is done and until sinal disease is discussed in terms of cytologic findings as well as in terms of other diagnostic procedures.

There is a definite correlation between the cell count of the secretions and the biochemical composition. Watery secretion is usually present when the discharge is profuse, and so the dilution is high and few cells may be seen. Sometimes secretion is scanty, and the flow may be started by the insertion of a tampon saturated with a solution of sodium chloride. Secretion may be obtained from various sinuses by washing. Sometimes centrifugation of the secretions is required. Full directions for making a smear and staining it are presented. Interpretation of the cytologic picture must include consideration of the type and quantity of the secretion and the correlation of this with the history and nasal changes.

There is no definite relationship between blood eosinophilia and clinical symptoms or the number of eosinophils in the nasal secretions.

Nasal allergy may persist without asthma, but often an allergic bronchitis appears, which is frequently a forerunner of asthma in children. Asthma may appear in severe form after acute infectious diseases. Whooping cough is the most common cause of its precipitation and measles is the next.

M. V. MILLER, Philadelphia.

THE USE OF HUMAN CONVALESCENT SCARLET FEVER SERUM IN STREPTOCOCCIC INFECTIONS INVOLVING THE EAR, NOSE AND THROAT. PIERRE VIOLE, Ann. Otol., Rhin. & Laryng. **49**:639 (Sept.) 1940.

Viole reports the result of using pooled scarlet fever serum in the treatment of hemolytic streptococcus infections. While the use of the serum does not give brilliant results in all cases, there seems to be definite evidence that it is of considerable value. This is particularly true in cases of streptococcic sore throat and cases of laryngotracheobronchitis. If given early enough and in sufficient quantity in the latter, the serum appears to eliminate the need for tracheotomy. Sulfanilamide does not appear to be needed in conjunction with it, but as long as they do not counteract each other, both should be used when indicated. There are no hazards in the use of the serum, and such reactions as occur are easily controlled.

M. V. MILLER, Philadelphia.

A SURVEY OF THE RELATION BETWEEN NUTRITION AND THE EAR. GRANT SELFRIDGE, Ann. Otol., Rhin. & Laryng. **49**:674 (Sept.) 1940.

Selfridge reviews the literature on deficiencies of the various vitamins and the effect of these deficiencies on the function of the eighth nerve and on the incidence and severity of infections. Lack of vitamin A, B or C apparently causes changes in the eighth nerve, in addition to other more general effects. Deficiencies in B and C appear to have a distinct bearing on certain allergic manifestations. All the vitamins are essential during acute infections; they do not shorten the infection but seem to aid in sustaining the general physical condition. The tendency to catch cold appears lessened if the diet contains all of the essential vitamin factors in sufficient quantity.

M. V. MILLER, Philadelphia.

ORAL POLLEN THERAPY IN RAGWEED POLLINOSIS: A COOPERATIVE STUDY. SAMUEL M. FEINBERG, FRANCIS L. FORAN, MEYER R. LICHTENSTEIN, EMANUEL PADNOS, BEN Z. RAPPAPORT, J. SHELDON and M. ZELLER, J. A. M. A. **115**:23 (July 6) 1940.

Feinberg and his co-workers conducted a study on patients suffering from ragweed pollinosis. Results in patients receiving oral pollen therapy were checked with those in an equal number receiving placebo capsules. These studies were carried out in the clinics of the University of Illinois College of Medicine, Rush Medical College, University of Chicago, the University of Michigan Medical School and Northwestern University Medical School.

Thirty-two patients received oral pollen therapy for uncomplicated ragweed hay fever with the following results: 18.75 per cent (6) were slightly improved; 18.75 per cent (6) had moderate to good results. Of the 32 patients receiving placebo capsules, 1 (3.1 per cent) was slightly improved, while 6 (18.75 per cent) had moderate to excellent results. Of 39 patients with asthma who received oral pollen therapy, 7 (18 per cent) stated that they had moderate to good results.

Two groups of children studied by different observers showed conflicting results. In one group, 11 patients receiving pollen orally reported uniformly favorable results. In another group of 15 receiving pollen orally, only 2 reported slight improvement.

The conclusions arrived at are substantially:

1. In their appraisal of oral pollen therapy made on the basis of their combined results, the opinions of the investigators diverge from a complete rejection to a limited acceptance.

2. Oral ingestion of pollen as used in the study reported is safe, despite the production of some gastrointestinal symptoms, but the therapeutic effectiveness is of minor grade, especially in asthma, and definitely inferior to that of parenteral injection of pollen extract.

GORDON, Philadelphia.

POSTOPERATIVE ATELECTASIS. LLOYD H. MOUSEL, J. A. M. A. **115**:899 (Sept. 14) 1940.

Mousel discusses the diagnosis and treatment of postoperative atelectasis from the point of view of the anesthetist. He is of the opinion that in most instances postoperative atelectasis is caused by mechanical obstruction, i. e., actual plugging of a bronchus or several bronchi by tenacious secretions, the latter being present preoperatively, during the operation or postoperatively.

Emphasis is placed on keeping the airway free and clear. Mousel comments on the preoperative details, the question of adequate sedation, indications for tracheobronchial aspiration and the technic thereof and the administration of carbon dioxide and oxygen in cases in which this is required.

The patient should be turned from side to side frequently, and he should be encouraged to cough and to breathe deeply.

Several case reports are presented, with roentgenograms and other data. Mousel concludes as follows:

"1. Much can be done by the anesthetist toward preventing postoperative atelectasis.

"2. Patients who have aspirated vomitus . . . mucus . . . blood . . . in the trachea at the termination of anesthesia should be submitted to tracheobronchial aspiration before leaving the operating table. The bronchoscope can usually be passed without difficulty if it is done before the patient has recovered from the anesthetic.

"3. Postoperative atelectasis can be successfully treated in most instances by bronchoscopic aspiration.

"4. Bronchoscopic aspiration should be carried out as soon as possible after the diagnosis is made in order to prevent pneumonitis or pulmonary suppuration."

GORDON, Philadelphia.

THE HYPERACTIVE CAROTID SINUS REFLEX SYNDROME. ARNOLD KOFFLER and STEWART F. ALEXANDER, New York State J. Med. **40**:1519 (Oct. 15) 1940.

Koffler and Alexander report 8 cases demonstrating the hyperactive carotid sinus reflex syndrome. The carotid sinus is innervated by one of the afferent branches of the autonomic nervous system, a nerve plexus situated near the bifurcation of the common carotid artery. Normally these nerves exert a tonic inhibitory influence on the heart rate and the systemic arterial tension. It is a protective mechanism to prevent embarrassment of the myocardium. Enlarged lymph nodes, tumors and other abnormal developments may irritate the end organs, which lie in the arterial walls; and a slight stimulus may cause syncope, particularly in the senile patient with calcareous deposits.

In regard to elderly patients with vertigo or convulsions, one may make a diagnosis of epilepsy, idiopathic syncope, cerebral arteriosclerosis, hysteria, Ménière's syndrome or syphilis of the central nervous system. Almost one-half the patients spoke of nausea and vomiting, which are probably due to general or mixed reflex action involving the viscera. The varied symptoms are due to a diffuse autonomic motor impulse pathway.

Treatment consists of general therapeutic measures, which are "of great value." Mild sedation with phenobarbital and the regulation of the daily regimen have given satisfactory results. Tight neckwear is to be avoided, as are sudden movements of the head and body. Denervation may be indicated in certain cases, but it was not performed in any of the 8 reported by Koffler and Alexander.

VOORHEES, New York.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY AND MILWAUKEE OTO-OPHTHALMIC SOCIETY

J. B. Hitz, M.D., *President of the Milwaukee Society*

March 11, 1941

Cavernous Hemangioma of the Nose, Nasal Septum and Forehead: Report of a Case. Dr. Samuel Salinger, Chicago.

A woman aged 20 was first seen in May 1937, for relief of an enlargement of the nose which had been present since birth. There was a smooth enlargement of the lower half of the nose, of bluish purple color, elastic to the touch and extending almost to the level of the lower border of the nasal bones. The lower portion of the septum bulged into the nares, causing almost complete obstruction. The mucous membrane was thin and pulsated slightly. The entire mass was easily compressible externally and internally. To the left of the midline of the forehead was a similar mass extending from the hair line toward the supraorbital margin. While the color was much like that of the nasal swelling, the mass felt somewhat more dense.

It was felt that surgical intervention was contraindicated, and the patient was referred to the Michael Reese tumor clinic, where interstitial irradiation was applied. As the septal portion of the growth remained practically unchanged, although there was reduction in the size of the nose, it was decided to treat it more directly by implantation of radium needles. After this was done, it was apparent in November 1939 that the vascularity of the masses had been greatly reduced, and plastic procedures were undertaken for cosmetic purposes.

In January 1940, the mass on the forehead was excised in toto with the overlying skin and the defect was closed by undermining the adjacent skin, which was brought together with tension sutures. This mass extended to the periosteum. The bulbous nose was reduced by two parallel excisions of elliptic wedges of skin and underlying stroma on each side of the nose from the level of the lower border of the nasal bones to within a few millimeters of the alar margins. The resulting scars were not entirely satisfactory and were resutured a short time later with much better cosmetic effect. A few pit marks will be excised at a future time.

Histologic examination of the tissue showed a large amount of fatty tissue irregularly distributed throughout the stroma, some muscle tissue and a large number of blood vessels showing various degrees of hyalinization. The vessel changes were undoubtedly due to the application of radium.

The case is reported because of the comparative rarity of the condition and to demonstrate the value of preliminary irradiation for reduction of the vascularity of an otherwise inoperable type of tumor. I feel that in this case the lesions were a developmental anomaly.

DISCUSSION

Dr. Harry Foerster, Milwaukee: I wish to compliment Dr. Salinger on obtaining an excellent cosmetic result in a difficult case. The condition in the case he presented is unique in some respects, as indicated by the histologic observations. It represents a developmental anomaly rather than a new growth. I recall a case Dr. Mulsow and I had at the county dispensary some years ago in which the patient had numerous vascular lesions involving both the skin and the mucous membranes and in which the pathologist reported the tissue removed as lipoma. The lesions were vascular tumors with an abundance of proliferative fatty tissue.

A cavernous angioma that is in the midline, in the location of this patient's lesions, may be due to developmental defects involving the deeper tissues and even extending to the meninges. This is something to bear in mind when employing surgical treatment for highly vascularized lesions in similar locations.

The surgical procedure is to be discussed by the next speaker, who has had much experience in this line. The advisability of not attempting to rely on one procedure but of using a combination of methods should be emphasized. There is no question that some angiomatous lesions can be satisfactorily removed surgically, and that many others are amenable to radiotherapy alone and the cosmetic results may be good. There are, however, certain cases in which the results with combined treatment, as cited in this case report, are far more satisfactory than those obtainable by either method alone.

Dr. Salinger spoke of pits in the skin which had to be corrected. This is an important point in connection with interstitial irradiation. Unless the physician is experienced he is likely to produce localized areas of atrophy in the skin because of the radium needles not having been planted deeply enough. As he said, implants of radon seeds are sometimes preferable because of this, but a difficulty with radon seeds is that they may move around in the loose vascular tissue after they have been planted, and if they get too close to one another necrosis may occur, while in other parts of the field, from which the seed has moved, inadequate effects of the irradiation necessitate further treatment.

The most satisfactory results in the treatment of cavernous angioma are obtained with irradiation in patients who are treated in infancy or early childhood. As the patient becomes older the angiomatous tissue becomes more mature; it is then less radiosensitive and therefore to obtain an effect one must depend on destruction of tissue by the radiation. In such cases an alternative treatment that has proved satisfactory, and that is to be recommended, is injection of a sclerosing solution, the most satisfactory being probably quinine hydrochloride and ethyl carbamate (urethane). This method likewise requires a certain amount of experience, but it does not require nearly so much as radium therapy, nor is there the same risk of necrosis. Permanent damage of the skin by irradiation is not infrequent, whereas the injection of a sclerosing solution, if carried out with proper technic, may produce very satisfactory results.

The remarks with reference to the ineffectiveness of superficial radium therapy in cases like the one discussed should be stressed by being repeated. Lesions like those in that case are not amenable to surface applications of radium because the dosage required to destroy the angioma is bound to damage the overlying skin irreparably.

Dr. George V. I. Brown, Milwaukee: I have selected a few pictures that illustrate some unusual and not generally understood features of angiomatous and similar growths. It would be more interesting if I could show colored motion pictures which I have, taken in some instances from two to four years after operative treatment, but I hope these still pictures may give a sufficient demonstration.

Dr. Salinger's interesting illustrations of the good results he has secured by combined treatment and operation are worthy of commendation, because in reconstructing the disfigured nose he has accomplished the necessary improvement by the use of immediately surrounding tissue. This I feel is important, because often a flap is transposed from a more or less distant part and, although the appearance of a nose so constructed may be satisfactory so far as outline is concerned, the tissue almost always continues to look as though it belongs somewhere else, not on the face.

One lantern slide shows an angioma which covered almost the entire side of the face, the nose, the lips and the lower eyelid of an infant; the prognosis seemed to be almost hopeless from an operative point of view, and yet the child recovered spontaneously. A second slide shows an angioma on the side of an infant's head and face. This grew to enormous size in eighteen days. After undergoing ligation

of both external carotid arteries without benefit, the child finally came for operation, the end result of which is effective, as shown. Such an angioma may continue without outward change from birth and in middle life begin to grow rapidly and require immediate operation. The method employed for removing a large angiomatous growth safely and effectively is illustrated.

A third slide shows a pigmented hairy mole covering the face of an infant, including both eyelids; most of the forehead and upper part of the face was cured by skin grafting. Part of the growth was left for an eyebrow. There were nine of these pigmented moles on this infant's body, and in the course of a few years all have disappeared spontaneously except one which is evidently disappearing. In contradistinction to this, other slides illustrate similar cases in which the pigmented surface had been excised and the whole skin graft placed but there was noticeable a slight tendency to recurrence of the pigment through the transplanted skin.

The purpose of showing these illustrations of growths which disappeared without treatment and also of some which without apparent difference in macroscopic appearance or microscopic examination grew rapidly is to bring out the little understood differences in the reaction, which probably explains the contradictory reports of the results of treatment by various methods.

DR. SAMUEL SALINGER, Chicago: I should like to say that I appreciate the discussion, and I also want to emphasize a point that Dr. Foerster mentioned, namely, that these congenital blood tumors are more successfully treated in infancy and in early childhood than when the patient reaches adult life. Also, there are so many different types of these tumors about the face, occurring at different levels and of different degrees of vascularity, that the treatment must necessarily vary according to the location, size and variety of the lesion. One therefore finds different agents being used in different cases. Surgical removal, surface irradiation, interstitial irradiation, electrodesiccation and application of solid carbon dioxide are all successful if intelligently applied. In many cases it will require the combined judgment of the surgeon, the dermatologist and the radiologist to determine what is best.

Early Diagnosis of Carcinoma of the Esophagus. DR. J. R. LINDSAY, Chicago.

Esophageal carcinoma, according to statistics, comprises about 4 per cent of malignant tumors.

The disease has been universally fatal. Radiation therapy has produced palliation only in occasional cases.

Surgical removal of esophageal carcinoma is now successfully carried out in 50 per cent of cases. Information gained through surgical exploration indicates that a cure through removal is possible in a moderate percentage of cases if diagnosis is made early.

Sixty cases in which carcinoma of the esophagus was treated in recent years at the University of Chicago Clinics are reviewed. In more than 50 per cent of these the diagnosis was not made until beyond four months after the initial complaint. The possibility exists, therefore, of making a diagnosis at least four months earlier in about half the cases. This should greatly increase the prospect for successful surgical removal.

Early diagnosis can be achieved only by means of roentgen and esophagoscopic examination in the presence of even mild complaints associated with swallowing. The three essentials for an esophagoscopic examination are: a thorough examination, including biopsy when indicated; the patient's safety, and the patient's comfort. The first two depend on the skill of the examiner, and the latter, on the preparation of the patient and the anesthesia. General anesthesia is preferable for

safety under certain conditions, but with care in preparation of the patient the operation may be done with local anesthesia without creating apprehension or distress during the procedure.

DISCUSSION

DR. JOHN GORDON, Milwaukee: Physicians who have had experience with malignant diseases of the esophagus know what the prognosis has been up to the present time. When one makes a diagnosis of malignant disease of the esophagus, one has to prepare oneself to sign the death warrant for the patient.

With the marked development of thoracic surgery in the past ten years, there seems to be much more to offer patients with malignant disease of the esophagus provided they can be seen in the early stages. Unfortunately, most physicians see such patients far too late. The thing that leads them to seek medical aid is not the early symptoms but the late ones, such as difficulty in swallowing soft foods and even inability to swallow fluids. Usually by the time these symptoms appear there has been such a degree of extension to the surrounding parts that surgical removal is almost impossible.

If otolaryngologists as a medical group are to help further the work of the thoracic surgeons in removing esophageal neoplasms, it will have to be done through a broadspread educational campaign to the laity and to the attending physicians, so that persons with esophageal symptoms will be seen at a very early stage.

Esophagoscopy many times will permit of a definite diagnosis much sooner than roentgenographic or fluoroscopic study will, and when there is any question as to lesion or diagnosis an esophageal examination should be made.

The men who are doing the newer types of esophageal resection for malignant disease deserve a great deal of credit. Up to the present time they have had little reward for their efforts. I believe with earlier diagnosis and earlier resections the mortality of esophageal malignant growths will be reduced.

Chronic Suppurative Otitis Media, Facial Paralysis, Circumscribed Meningitis and Acute Labyrinthitis: Report of a Case. DR. HOWARD C. BALLENGER, Chicago.

The report of this case is to be published in full in a future issue of the *Annals of Otology, Rhinology and Laryngology.*

DISCUSSION

DR. T. L. TOLAN, Milwaukee: I think it is encouraging in these times of sulfanilamide and its derivatives to know that patients such as the one Dr. Ballenger has reported on got well even before the new therapy. It seems impossible at this time to feel that such patients would recover unless we could give sulfanilamide credit for the cure. It is particularly interesting that the patient had paralysis of the seventh nerve before operation and that paralysis of the sixth nerve developed after the operation.

The case is interesting, and I think Dr. Ballenger is to be congratulated in pursuing a watchful waiting attitude, after thorough exenteration of the mastoid.

DR. ALFRED LEWY, Chicago: In Dr. Ballenger's case there was no positive bacterial culture of the spinal fluid, indicating that the condition was not a generalized meningitis but probably secondary to the local infection. My diagnosis would be that it was a chronic mastoiditis with petrositis probably, and that the other intracranial symptoms, paralysis of the seventh and sixth nerves, were due to edema of the brain. I have a patient with a similar condition in the hospital now. Before the introduction of sulfanilamide there was usually recovery from meningitis with negative cultures but not from meningitis with positive cultures.

My patient is a Negro boy who should have been operated on last December when I first saw him. He came in a few days ago with a full-blown meningitis. There was still some hearing left in the ear, and the usual radical operation was

performed, with uncovering of the dura in both the middle and the posterior fossa. The dura was changed in color, not thickened and not under pressure. The day after the operation the boy had complete aphasia and complete hemiplegia on the right side. The neurologist's idea was that there was an abscess of the sphenotemporal lobe. I thought that such an abscess could not have developed in so short a time. On the second day the boy recovered movement of the right arm and leg but was still aphasic. He is now recovering his speech. I hope he recovers. I do not think he had an abscess but the neurologist insists that he did.

DR. SAMUEL SALINGER, Chicago: Dr. Ballenger brought up a point on which I should like to have him elaborate, namely, the question as to whether the labyrinthitis was secondary to the meningitis or the meningitis secondary to the labyrinthitis. This has been mentioned in the literature and was definitely proved by Ruttin in a case in which autopsy showed that labyrinthitis was due to retrograde infection through the internal auditory canal from meningitis. In certain cases the indications for treatment might depend on having this information. I should like to have Dr. Ballenger give his reasons for his conclusions.

DR. HOWARD BALLENGER, Chicago: I want to thank Dr. Tolan for his discussion. I am glad to have Dr. Lewy's analysis of this case; however, petrositis with edema of the brain would not account for the spinal fluid cell count of 1,100. Some intracranial infection was present. Roentgen examination showed nothing except chronic mastoiditis. So far as I know there was no evidence of petrositis.

In reply to Dr. Salinger's question, the symptoms of meningitis were present before the patient was admitted to the hospital. The symptoms of labyrinthitis developed later. For that reason I assume if an infection did occur in the labyrinth it was by way of the internal auditory meatus.

Kodachrome Motion Picture Studies of Pathologic Conditions of the Larynx. DR. PAUL HOLINGER, Chicago.

(The color motion picture presented illustrated various types of laryngeal pathologic conditions as seen through the direct laryngoscope. Acute and chronic inflammatory lesions and laryngeal neoplasms were shown. The author's equipment for laryngeal photography was described.)

DISCUSSION

DR. HERBERT SCHMIDT, Milwaukee: These moving pictures of the larynx speak for themselves. The work is so beautifully done that it appears to be a simple matter, but to take such pictures requires a high degree of skill in photography. Certainly all of us present will agree that this is an ideal way of showing conditions in the larynx, even the normal larynx. For teaching purposes it will be invaluable. In one evening a student or a physician may see lesions or conditions which could otherwise be seen only over a long period of time if he had to wait until patients could be observed in the clinic.

I presume that in the future Dr. Holinger will be able to take photographs in many more interesting cases that come to his attention and will probably show us more and more of these laryngeal conditions.

DR. WALTER THEOBALD, Chicago: I should like to comment on the necessity of the type of work that is being done by Dr. Holinger at the bronchoscopic clinic at the University of Illinois, at St. Luke's Hospital and at Children's Memorial Hospital. The importance of direct laryngoscopic examination became apparent to me in the case of a man who had had hoarseness for six months. On one side there was a very marked bulging red infiltration of the larynx beginning at the right false cord and extending onto the true cord, which evidently was subglottic. Also, there was a slight deviation of the larynx and trachea to the left side at this point, as shown by roentgen examination. With this long history of hoarseness, I could think only of a malignant growth. To make a thorough and exhaustive study, and to be sure the process was not malignant, I recommended exami-

nation by Dr. Holinger. The entire infiltration was found to be perfectly smooth. However, a growth was seen extending toward the trachea from the thyroid gland. I feel sure that this patient does not have a malignant growth but perhaps has a thyroid growth outside the trachea. The basal metabolic rate shows a range of +50 to +29 per cent. The patient is now being given potassium iodide and later will probably have an operation on the thyroid. The importance of being sure one is not dealing with a malignant growth is shown by this case, and one can be sure only through direct examination.

Dr. John Mulsow, Milwaukee: I have often thought that if it were possible it would be interesting in this type of photography if one could insert the lateral roentgenogram to show the amount of invasion of surrounding tissue by the tumor, which is not seen in direct or indirect laryngoscopic examination.

With reference to Dr. Theobald's case, some time ago I saw a patient who had considerable difficulty in breathing, with evidence of pressure on the trachea, and roentgen examination showed a large substernal goiter.

Book Reviews

Essays on the Applied Physiology of the Nose. By Arthur W. Proetz, M.D., Professor of Clinical Otolaryngology in the Washington University School of Medicine, St. Louis. Price, $7. Pp. 395, with 91 illustrations, St. Louis: Annals Publishing Company, 1941.

A new textbook on the applied physiology of the nose is presented to the medical profession by Dr. Arthur W. Proetz. It constitutes the first text of its kind and represents the latest contribution of this distinguished rhinologist to American otolaryngology.

The book is built on strong pillars. Fundamental principles based on the author's laboratory and clinical studies, together with the closest collaboration with men outstanding in the fields of otolaryngology, anatomy, cytology and physiology; knowledge, judgment, depth of understanding, reflection and maturity of thought arrived at after many years of teaching and discussions with eminent rhinologists; exchange of views with others during the meetings of the various national and local laryngologic bodies; the rich background of the author and his constant endeavor to be exact, precise and certain and to leave controversial subjects to be revised at the proper time; the zeal of the author in combing the literature to make his theme as complete as possible; the lucidity and clarity of his writings—these and other attributes impart strength and truth to this volume.

Much is compressed in 395 pages. The twenty-one chapters include historical notes, discussions on the functions of the nose, structure as a basis for function, the sense of smell, the rigidity of the nasal structure, air currents in the nose, air pressure, respiration and air exchange, humidification, heating, electrical surface charges, cilia (morphology, action, growth and regeneration and physical and chemical reactions), other defenses of the mucosa, vascular and lymphatic reactions, neural reactions, climate, environment and other extrinsic influences, clinical assets of nasal treatment and clinical aspects of nasal surgery, and there is an appendix of 14 pages, with instructions for performing some experiments illustrating sinus drainage, etc.

The bibliography for each section is complete, and, in addition, supplementary references are given to allow the reader the widest latitude for studying the subject to the fullest extent.

This textbook is recommended to otolaryngologists, graduate students in otolaryngology and residents and clinicians working in collaboration with otolaryngologists. It should be particularly useful for those preparing for certification by the American Board of Otolaryngology.

Dr. Proetz should be congratulated for his splendid contribution and merits the gratitude of the medical profession in general and of the otolaryngologic world in particular.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank R. Spencer, Physicians' Bldg., Boulder, Colo.
President-Elect: Dr. Ralph I. Lloyd, 14-8th Ave., Brooklyn.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 19-24, 1941.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. T. E. Carmody, 227-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western.—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.